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THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 52 DECEMBER, 1944 No. 6

DILATATION OF THE VERTEBRAL CANAL ASSOCIATED WITH CONGENITAL ANOMALIES OF THE SPINAL CORD

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INTRODUCTION

THAT the spinal canal may be markedly dilated as the result of an intraspinal neoplasm is well known, but that an equally pronounced dilatation may occur at any level as the result of a congenital anomaly of the cord structures is not generally recognized. The present report concerns 3 cases of dilatation of the spinal canal associated with myelodysplasia, and a fourth case with a vascular anomaly of the spinal cord.

CASE REPORTS

CASE 1. *Male, aged twenty-five, complaining of a deformed left leg, paresthesias in saddle region, and urinary incontinence: atrophic left leg, bilateral sacral hypesthesia and neurogenic bladder; dilatation of sacral canal demonstrated by roentgenograms and at operation with lipoma of conus medullaris adherent to coccyx: marked improvement after removal of lipoma.*

J. S. (Unit No. 218520), a steel worker, aged twenty-five, was referred to the neurosurgical service by Dr. Charles Huggins on May 22, 1939. At the age of seven, the patient injured the left foot following which he noticed that the left leg was weaker than the right. Eight or nine years before admission a numbness developed over both buttocks. In the summer of 1936 he was first troubled with enuresis and urinary frequency. This progressed so that during the few months prior to admission he had to use a rubber condom at night. He experienced an urgent desire to micturate every two to three hours during the day.

On physical examination the patient appeared to be well developed except for the lower extremities. The heart and lungs presented no abnormalities. His blood pressure was 122/64. The left thigh was definitely smaller than the right, measuring 42 cm. in circumference, 15 cm. above the patella, as compared to 49 cm. on the right. The right leg, 15 cm. below the patella, was 33 cm. in circumference and the left 23 cm. The right knee and ankle jerks were livelier than those on the left. The plantar reflexes were flexor. In the saddle region below the fifth lumbar segment the appreciation of pain was impaired although the patient was able to distinguish between the head and the point of a pin. After voluntary micturition there was between 240 and 400 cc. of residual urine. Blood examinations including the Wassermann test were normal. Repeated urinalyses showed no abnormality other than the presence of numerous white blood corpuscles. A cystometrogram was interpreted as indicating a hypertonic bladder.

A lumbar puncture was performed. The initial pressure was 160 mm. of spinal fluid and the dynamics were normal. The fluid was clear and colorless. The Wassermann test was nega-

FIG. 1. Case 1. Roentgenogram of the lumbosacral spine showing the enlargement of the spinal canal in the sacral region and the spina bifida occulta of the first sacral vertebra. The interpedicular distances measured: thoracic 10, 21 mm.; 11, 21 mm.; 12, 23 mm.; lumbar 1, 27 mm.; 2, 28 mm., 3, 29 mm.; 4, 31 mm., 5, 33 mm.; and sacral 1, 42 mm.

tive and the Lange curve was 0000000000. The total protein of the spinal fluid was 39 mg. per 100 cc. Roentgenograms of the abdomen and pelvis revealed no abnormality in the soft tissue but an incomplete fusion of the laminae of the first sacral vertebra (Fig. 1). Cystograms showed marked trabeculation of the bladder and reflux filling of the right ureter. A pneumomyelogram was made which revealed a marked enlargement of the sacral sac. The history and findings suggested that the patient had a congenital abnormality of the lower part of the spinal cord.

On May 29, 1939, the patient was anesthetized with ether and a laminectomy performed in the lower sacral region. A midline incision was carried from the third lumbar spine to the lower border of the first sacral spine. The paravertebral muscles were separated by subperiosteal dissection from the spines, which were then removed. The laminae of the fourth and fifth lumbar and the first sacral vertebrae were then rongeured away. The dural sac appeared abnormally wide. When incised and the dural margins separated, the spinal cord was seen to pass down the center of the canal. The arachnoid over this was definitely milky and in places adherent to the dura mater. After lengthening the skin incision, the spines and laminae of the sacral vertebrae were removed. This exposed an enormous dilatation of the inferior portion of the spinal canal. It measured 4 cm. in width and was about 4 cm. anteroposteriorly. The spinal cord was seen to pass down the middle of this enlarged canal and terminate to the left of a mass of fatty tissue which was adherent to the end of the sacral sac. The lipomatous mass measured 2 cm. in diameter and from it numerous connective tissue trabeculae passed to the tip of the sacral sac. The roots of the sacral nerves passed from the spinal cord asymmetrically and ran slightly caudad or cephalad to the intervertebral foramina. The lowest roots on each side appeared to pass from the anterior surface of the fatty mass and enter the sacral foramina. The fatty mass was dissected and removed piecemeal to avoid damage to nerve fibers passing through it. The numerous fibrous bands attached to the dura were finally broken and the spinal cord allowed to retract in the spinal canal. This released the tension on the lower sacral roots. Each time the fatty mass was grasped with the tissue forceps the spinal cord was seen to tighten spasmodically and then relax. This twitching was seen several times and was considered to be due to the presence of muscle fibers in the lipoma or in its attachment to the dura. After careful scrutiny for bleeding points, the dura mater was sutured and the muscles and skin were closed in anatomical layers (Fig. 2).

Pathology Report. Sections of the tissue removed at operation showed it to be composed chiefly of large fat cells in a connective tissue stroma.

The patient had an uneventful convalescence although he required an indwelling catheter for almost two weeks after operation. The residual

urine decreased to 30 cc. but still contained many pus cells. On June 16 he was discharged from the hospital. The wound had healed *per primam*.

He was seen on several occasions following his discharge, the last one being November 17, 1939. He stated that he felt fine, was working regularly and had incontinence only occasionally while at work and never at night. The motor and sensory status of his lower extremities was essentially the same as preoperatively. The patient considered himself very markedly improved.

Comment. The caudal portion of the spinal cord is the usual part to be involved by anomalies. A diffuse lipoma infiltrating the roots of the cauda equina and extending through the laminae to the subcutaneous tissues presenting a mass over the sacrum covered by hairy skin is commonly seen associated with spina bifida occulta. The myelodysplastic condition seen in the present case, consisting of a single trunk from which the lumbar and sacral nerves are given off, instead of a cauda equina, is less frequently encountered. The trunk may be the site of various pathological processes such as lipomata, dermoid cysts, or adhesive bands[16] which may or may not be connected anatomically to the defect in the spinal lamina. The sacral canal is usually but not necessarily dilated.[21] A severe myelodysplasia may be present without dilatation of the sacral canal and with a normal appearing cauda equina.

The history of the present case is typical of myelodysplasia associated with spina bifida occulta. A mild impairment of function in one lower extremity noticed in early life followed by urinary disturbances beginning in adolescence and increasing in severity in early adult life is the story frequently given by patients with this disorder. The late occurrence of symptoms is probably related to the tension placed upon the spinal cord by the greater growth of the vertebral column. The relief of symptoms obtained by sectioning the adhesions binding the conus medullaris to the sacral sac seems to support this hypothesis.

Case II. *Male, aged thirteen months, suffering from a weak right leg since birth; spastic atrophic right leg with absent tendon reflexes; no sensory disturbance; enlarged lumbar spinal canal by roentgenography; marked myelodysplasia at operation.*

E. B. (Unit No. 284522), male, aged thirteen months, was referred to the neurosurgical service by Dr. Howard Hatcher on June 17, 1942.

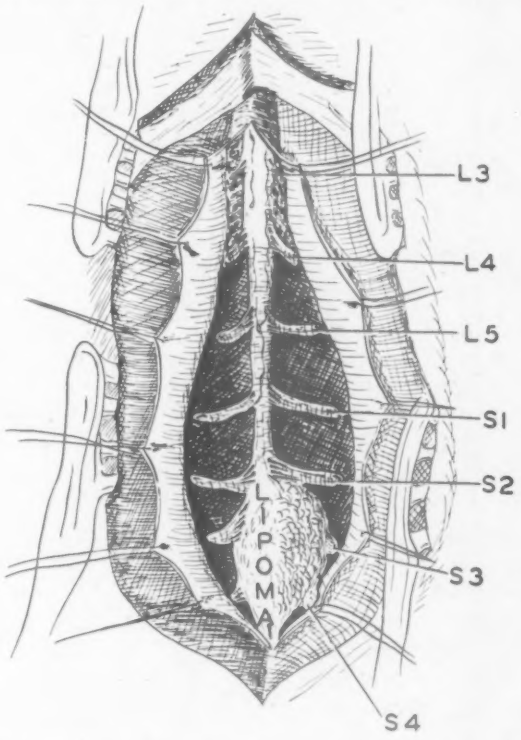

Fig. 2. Case I. Sketch of operative field to show the appearance of the caudal sac and its contents. The irregular spacing of the sacral roots which come off at right angles from the elongated conus medullaris is evident.

The parents had noted shortly after his birth that the baby did not move the right leg well. The limb seemed rather flabby and limp. The right foot appeared smaller than the left. When the child started to crawl, some improvement was noted but when he attempted to stand the right foot was everted. The birth history was not unusual, in fact his mother believed that the delivery was normal. Except for the presenting complaint his development was normal.

On physical examination the patient appeared to be a well nourished baby of about one year. The chest and abdomen presented no abnormality. The cranial nerves functioned well

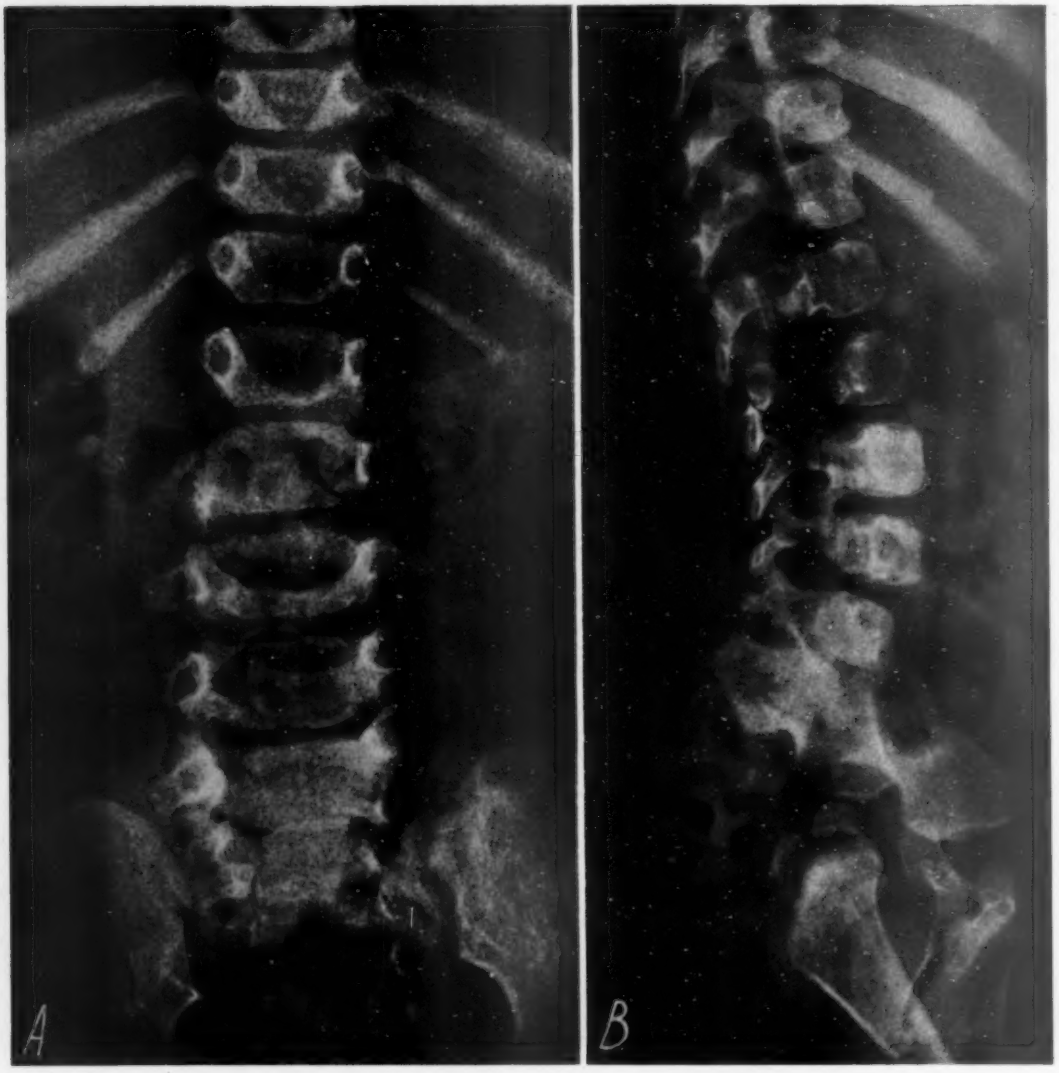

FIG. 3. Case II. Roentgenograms of the lumbosacral spine showing the marked dilatation of the spinal canal, and the anomalies of the laminae of the lumbar vertebrae. *A*, anteroposterior view; *B*, lateral view. The interpedicular distances measured: thoracic 6–11, 15 mm.; 12, 16 mm.; lumbar 1, 17 mm.; 2, 23 mm.; 3 and 4, 24 mm.; 5, 23 mm.; and sacral 1, 24 mm.; 2, 17 mm.

and the upper extremities were well developed and strong. The right leg was shorter and smaller than the left. The right calf measured 6 cm. in circumference and the left 6.5 cm. The right leg was spastic to passive manipulation but its tendon reflexes could not be obtained. The left leg had normal tone on passive manipulation although the left knee and ankle jerks were hyperactive. The left leg was moved freely at all joints. The right hip and knee could be flexed and extended fairly well but the right ankle could be moved very little. Sensation could not be accurately determined but no severe defect appeared to exist.

Roentgenograms of the lumbar spine revealed a marked increase in the width and depth of the spinal canal in the upper lumbar region. The right pedicle of the second lumbar vertebra was very thin and small. Its inferior half appeared to be missing. The laminae of the lower lumbar vertebrae were not fused (Fig. 3).

Examinations of the blood and urine revealed no abnormalities. The child was thought to have a congenital cyst of the spinal cord.

On June 18, 1942, the patient was anesthetized with ether and the lumbar region explored. A midline incision was made over the spines of the lumbar vertebrae and the muscles were then separated by subperiosteal dissection from both sides of the spines. The spines were very short and the laminae came off the spines almost immediately. The spines and laminae were rongeured away on both sides. There was little epidural fat and the spinal canal was unduly wide. The dura mater was incised longitudinally exposing the arachnoid and spinal cord (Fig. 4). The spinal cord was split into two cords, a larger one on the left side from which the nerve roots came off quite normally. On the right side, however, the nerve roots were irregularly spaced and came off of a smaller cord of tissue at right angles. A strand from the larger cord joined the smaller at approximately the level of the lower border of the third lumbar vertebra and a second bridge crossed just below this point. Below this level the two spinal cords were again fused. Since no surgical procedure was indicated, the dura mater was closed and the muscles approximated.

The patient had an uneventful convalescence and was discharged on June 28, 1942. He returned to the clinic on July 27, at which time the parents stated that his condition had changed very little. He was able to stand and take a few steps. The wound was well healed. The tendon reflexes were absent in the right but quite active in the left leg.

Comment. This case illustrates a dilatation of the spinal canal due to a diplomyelia or more probably a diastematomyelia. The neurological disturbances are probably the result of the myelodysplasia rather than being due to the fixation of the spinal cord abnormally low in the spinal canal.

The severe forms of myelodysplasia resulting in a doubling or splitting of the spinal cord have been usually a concomitant of an extensive spina bifida with meningocele, meningomyelocele or myelocele.[5,6,22] In those cases in which the vertebral canal has been well formed in the presence of diplomyelia or diastematomyelia, a dilatation of the canal has usually been present with or without a rudimentary division of the canal. Herren and Edwards[13] state that the spinal canal may show an abnormal appearance varying from a moderate degree of dilatation at the level of the duplication of the cord to partial doubling. In about one quarter of the cases they reviewed, osseous or cartilaginous processes were reported protruding from the dorsal surface of one or more vertebral bodies, partially dividing the spinal canal in two parts.

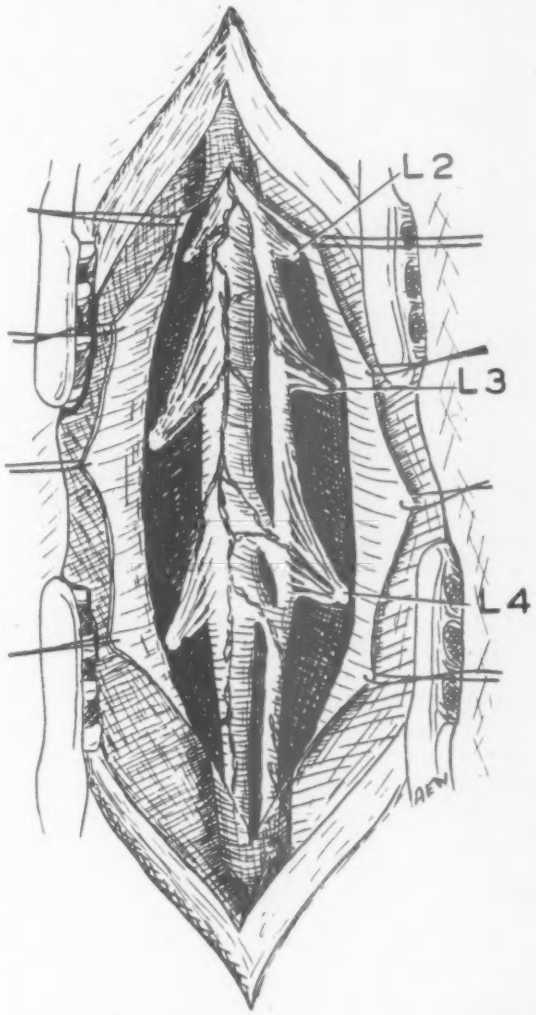

Fig. 4. Case II. Sketch of operative field to show the splitting of the spinal cord and other anomalies.

Case III. *Girl, aged four, following a fall developed a stiff painful neck and progressive weakness of the arms and legs; head traction gave some relief; marked dilatation of the cervical spinal canal; operation revealed a hydromyelia and diastematomyelia; autopsy.*

FIG. 5. Case III. Anteroposterior roentgenograms of the cervicothoracic spine showing the marked dilatation of the lower cervical and upper thoracic part of the spinal canal. The interpedicular distances measured: cervical 2, 28 mm.; 3 and 4, 29 mm.; 5, 31 mm.; 6, 34 mm.; 7, 37 mm.; thoracic 1, 39 mm.; 2, 35 mm.; 3, 29 mm.; 4, 26 mm.; 5, 22 mm.

M. L. M.,* a girl aged four and one-half, was admitted to the Bobs Roberts Hospital on November 27, 1937. Three months previously she had fallen, following which she developed a stiff and painful neck. She was suspected of having acute anterior poliomyelitis and sent to the Municipal Contagious Hospital where the diagnosis was not confirmed and the child sent home. She was symptomless for three weeks and then began to suffer headaches and vomited several times. After one week of these symptoms she was again sent to the contagious hospital. She was found to have weakness of both arms and legs, and a tendency to hold the head stiffly to the left side. A spinal puncture was said to have shown no abnormality in the spinal fluid. After two weeks she was transferred to the Children's Memorial Hospital where she was seen by Dr. Douglas N. Buchanan. Dr. Buchanan noted rotation of the head so that the chin rested on the right shoulder, gross weakness of both arms, more on the right side than on the left and more marked in the upper arm than in the lower, absence of all tendon reflexes, extensor plantar reflexes and a sensory level at the fourth cervical dermatome for pin prick. The patient was placed in head traction with relief of pain. A spinal puncture showed the fluid to be under normal pressure, and a good rise was obtained on jugular compression. There were three cells per cubic millimeter of spinal fluid and the Wassermann test on the fluid was negative. The tuberculin reaction was negative.

On November 4 the traction was removed but the patient's condition became worse. The child failed to improve and was transferred to Bobs Roberts Hospital for operation.

At the time of admission the child appeared acutely ill, although the heart, lungs and abdomen were not abnormal to the usual methods of examination. There was a complete motor and sensory paralysis below the second cervical dermatome. All deep tendon and abdominal reflexes were absent. The plantar reflexes were extensor. There was a marked vasodilatation of the capillary bed of the hand but the palms were red and mottled. Crutchfield tongs were applied the day of admission following which the patient appeared to be much more comfortable. The following day a lumbar puncture was performed. The initial pressure was 230 mm. of spinal fluid and there was a good rise on jugular compression. Four cells were present per cubic millimeter of spinal fluid and Pandy's test was negative.

Roentgenograms of the cervical spine showed a marked dilatation of the spinal canal from as high as could be seen to the fifth thoracic vertebra. The dilatation was widest at about the first thoracic vertebra where the canal measured approximately 4 cm. in width (Fig. 5).

On December 2, 1937 the patient was anesthetized with ether. With a preoperative diagnosis of congenital tumor of the spinal cord, a cervical laminectomy was performed by Dr. Paul C. Bucy. A midline incision was made from the suboccipital region down to the fifth thoracic spine. The arch of the atlas, spine and laminae of the second cervical vertebra ap-

* This case was reported briefly by Dr. Paul C. Bucy before the Chicago Pathological Society (Bucy, P. C. Gross congenital malformation of the spinal cord in a child five years of age. *Proc. Inst. Med., Chicago*, 1938, *12*, 81).

peared to be normal. From that point downward, however, to the fourth thoracic spine, the vertebrae had no spinous processes and the laminae failed to unite at the midline. The small defect between the ends of the laminae was bridged with fibrous tissue. From above downward and from the fourth thoracic vertebra upward, the laminae grew progressively longer until they were longest and the spinal canal accordingly widest at the level of the seventh cervical and first thoracic vertebra. The entire cervical and upper four thoracic laminae were resected. The dura mater was under considerable tension and did not pulsate until the arch of the atlas was removed. It presented a fusiform swelling conforming to the configuration of the spinal canal. The dura mater was incised longitudinally in the midline. It was very adherent to the underlying arachnoid membrane to which it was connected by numerous threadlike fibrous bands. At one point two large veins united at the midline on either side and passed from the arachnoid backward to the dura mater. Beneath the arachnoid could be seen a large collection of fluid extending into the caudal half of the exposed field where the arachnoid passed over and was in intimate contact with a much dilated spinal cord. In the upper half of the exposed field the spinal cord could not be seen. The arachnoid was incised at the midline. It contained many small vessels which were cauterized. On incising the arachnoid a large cystic cavity presented which did not appear to communicate with the subarachnoid space. This cavity extended from the base of the skull to the caudal limits of the exposed field measuring 8 cm. in length and 3 cm. in width. It was divided into two parts. In the upper half it consisted of a cavity closed on its posterolateral surface by a membrane which appeared to be the arachnoid.

The anterior wall of the cavity in this region was formed by very much flattened spinal cord covered with numerous brownish tufts of choroid plexus. In the middle of the exposed field the spinal cord was seen to split in half, each half migrating to the sides of the spinal canal leaving a cavity between the two halves. Caudally the two parts of the spinal cord reunited at the level of the fifth thoracic vertebra to form a single spinal cord. The cavity was continued into the central canal of the spinal cord for an indeterminate distance. The fluid in the cavity and in the subarachnoid space appeared to be identical. The arachnoid-like tissue forming the roof and the lateral walls of the cystic cavity was resected, opening a communication between the cavity and the subarachnoid space. The choroid plexus was not disturbed because its blood supply seemed identical with that of the spinal cord. The wound was carefully dried, the dura sutured, the bone fragments were laid along the margins of the laminae and the muscles and fascia were closed with two layers of deknatel and the subcutaneous tissues of the skin with two layers of interrupted silk.

The tissue removed from the wall of the cyst at the time of operation was composed primarily of connective tissue similar to that seen in the leptomeninges but was markedly thickened, very vascular and quite cellular. Numerous small blood vessels were present with many polymorphonuclear leukocytes in the vessels. On the inner surface of the tissue were ependymal-like cells and in some places blood vessels lined with cells characteristic of choroid plexus.

The patient did poorly after operation, however, and died approximately twelve hours later. Permission for autopsy was given and was carried out at 5:45 A.M. by Dr. W. A. Stryker. The diagnoses were bilateral pulmonary hyperemia and edema, hyperplasia of the mesenteric lymph nodes, aberrant pancreatic nodules and degeneration of the liver, kidney and myocardium and hydromyelia and myelodysplasia.

Examination of the Central Nervous System. Gross: Beneath the dura mater was a large cyst enclosed by an arachnoid-like membrane. This cyst was separate from the subarachnoid space. From the level of the first cervical to the fifth cervical vertebra the cyst lay posterior to the spinal cord which was flattened and formed its anterior wall. On the dorsal surface of the spinal cord at this level were many brown papillomatous structures, grossly considered to be masses of choroid plexus. At the level of the sixth cervical vertebra the dorsal surface of the spinal cord split leaving a large opening through which the cystic cavity passed. At this level the spinal cord consisted of two large lateral masses which surrounded the cystic cavity and were united anteriorly by a thin sheet of tissue. At about the level of the fourth thoracic vertebra the two halves of the spinal cord united again, leaving a hydromyelic cavity in the center of the cord (Fig. 6).

Microscopic: Sections taken at fairly uniform intervals from the spinal cord and stained by Nissl's and Weil's methods and hemotoxy-

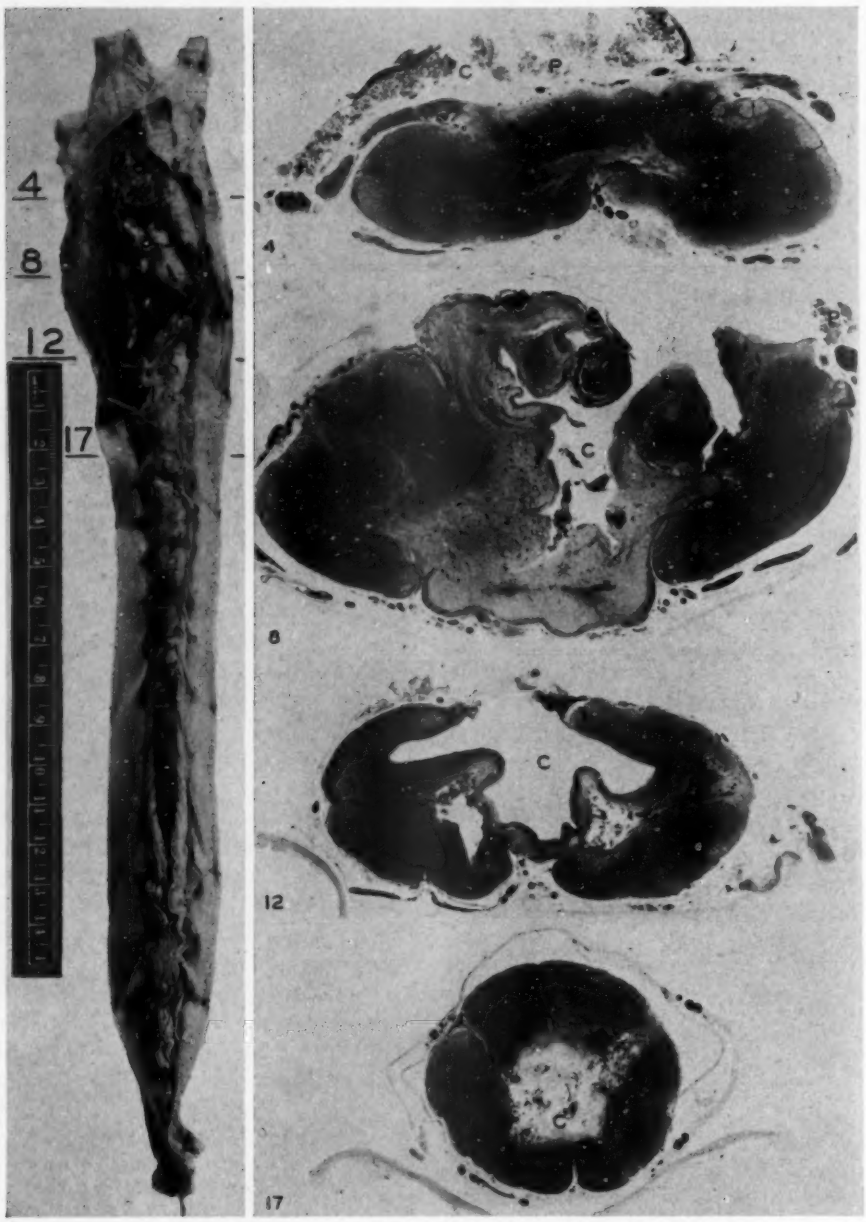

Fig. 6. Case III. Photograph of the spinal cord (dorsal surface) showing the anomaly in the cervical region (left) and photomicrographs of four sections of the maldeveloped spinal cord at different levels. The lines at the sides of the spinal cord indicate the site of the four sections from above downward respectively. *C*, cyst; *P*, choroid plexus.

lin and eosin show that the malformation consisted of a cystic posterior dilatation of the central canal of the spinal cord in the cervical region with the formation of a cavity in which choroid plexus was differentiated. In the lower portion of the spinal cord the central canal was well formed, slightly dilated and filled with debris. In the upper thoracic regions, its structure was broken up and only fragments of the walls were present. The central gray matter was likewise disintegrated, although the anterior horn cells were readily distinguished on each side. Slightly higher the destruction of the central gray matter was more prominent and

apparently traumatic for considerable blood was present. The dilated central canal could not be distinguished although fragments of ependymal columns were scattered about. Slightly higher the disintegrated central area reached the surface posteriorly. Above this level the walls of the posterior surface of the spinal cord were fragmentarily covered with cuboidal ependymal-like cells. These seemed to join with the central canal which at this level split in two. Higher the cavity lost its continuity with the central canal, becoming more and more peripherally situated and containing tufts of choroid plexus arising from the cuboidal lining. Two anterior fissures were present at this level on either side of which were anterior horn cells and between which lay a mass of nerve fibers (anterior commissure). In the upper cervical region the normal anatomical structure of the spinal cord was reformed, but lying on its dorsal surface adherent to the arachnoid was the cuboidal lining of the cystic cavity and a few fragments of choroid plexus. Above the level of the cyst the spinal cord appeared practically normal (Fig. 6).

Comment. Splitting of the spinal cord in the cervical region is rare; only two examples in this region are reported and neither lived after birth.[1,11] The great majority of such cases occur below the mid-thoracic region. The essential difference between the present case and those occurring in the lower portion of the spinal axis is the presence of choroid plexus in the cystic cavity. In many other respects this anomaly resembles the malformation described by Weil and Matthews.[20] The inclusion of choroid plexus presumably indicates that the dysraphism involved anlage of structures taking part in the formation of the fourth ventricle.

Case IV. *Male, aged fifteen, stricken with abdominal pain; previous history of deformity of left foot diagnosed anterior poliomyelitis, followed by urinary incontinence; marked atrophy of legs with absent tendon reflexes; bloody spinal fluid; marked widening of lower thoracic and lumbar spinal canal; angioma and aneurysm of spinal cord at operation; slow recovery.*

F. K. (Unit No. 276616), male, aged fifteen, was referred to the neurosurgical service by Dr. Howard Hatcher on December 25, 1941. His mother stated that he seemed well until the previous evening when she found him crying with abdominal pain. He stated that he had been suddenly stricken with pain in his side, had fainted, and fallen to the floor striking his head. The boy was able to walk and talk coherently to his mother. He vomited at supper that evening and refused everything by mouth on the next day.

The patient was a full term baby, delivered normally, who developed well until about the age of seven or eight years when he began to walk with a peculiar gait. His left foot rotated inwardly and he slowly developed a valgus deformity of the left foot. Although the onset of this trouble was not acute, it was diagnosed as an anterior poliomyelitis and was treated with casts and later by a tendon-splitting operation. At about the same time the patient developed enuresis and urinary incontinence so that he was seldom continent for more than two or three days at a time. On August 22, 1941, his urine was bloody and remained so for several weeks. From that time he became weaker, paler and lost weight.

At the time of admission he was irritable, lethargic and exhibited generalized muscular rigidity. On examination he had an obvious squint which was congenital. His blood pressure was 130/80 and there were no abnormalities in the cranial nerves. The heart and lungs appeared normal. The urinary bladder was markedly distended and the abdomen was quite rigid. The left leg was thin and wasted. There was a bilateral clawfoot, the left more marked than the right. The legs were spastic but the strength was fairly good. There was a sensory level with relative hypalgesia below the twelfth dorsal segment. The tendon reflexes in the lower extremity were absent. The left plantar response was extensor. The neck was retracted and Kernig's and Lasègue's signs were positive.

Roentgenograms of the lower thoracic and lumbar spine demonstrated a marked widening of the spinal canal with erosion of the pedicles of the twelfth thoracic and first lumbar vertebrae and some erosion of the pedicles of the second lumbar vertebra (Fig. 7). A spinal puncture was performed on December 26, 1941. The fluid was definitely serosanguineous containing many red and white blood cells. Supernatant fluid after centrifuging was xanthochromic. The lumbar puncture was repeated the following day and again bloody spinal fluid was

FIG. 7. Case IV. Anteroposterior roentgenogram of the spinal column showing the narrowing of the pedicles and dilatation of the spinal canal in the upper lumbar region. The interpedicular distances measured: thoracic 6, 18 mm.; 7, 19 mm.; 8, 20 mm.; 9, 22 mm.; 10, 25 mm.; 11, 26 mm.; 12, 33 mm.; lumbar 1, 40 mm.; 2, 33 mm.; 3 and 4, 30 mm.; 5, 33 mm.

removed. The dynamics could not be determined due to the uncooperativeness of the individual.

On January 8, 1942, the patient was anesthetized in the lower thoracic region by infiltration of 1 per cent novocaine into the skin and paravertebral muscles. A midline incision was carried from the tenth thoracic to the third lumbar vertebral spine. The muscles were separated from either side of the spines. It was apparent that the spines of the twelfth dorsal and first lumbar vertebrae were very short, measuring not more than 1.5 cm. in length and that the laminae at these levels were quite wide, long and not more than 1 or 2 mm. in thickness. The spinal canal was accordingly markedly dilated. The laminae of the twelfth thoracic, first and second lumbar vertebrae were rongeured away exposing a thick dura mater with thickened epidural fat. At the upper margin of the twelfth lumbar vertebra the epidural fat was much more normal and the spinal cord was seen to approach a normal size. Below this level the canal markedly widened to the level of the second lumbar vertebra where it again became smaller. The dura was incised in the midline. Instead of the normal spinal cord, a mass presented, completely filling the canal. One nerve root could be seen leaving this mass and passing across a large sclerotic blood vessel. The mass, yellow in color, appeared to be continuous with the normal spinal cord above and below. The right side of this enlarged spinal cord was covered with tangled fine blood vessels. On the left side there appeared to be a bluish colored cystic area. Upon incising the latter, a little clot of blood was encountered. The right side was then incised and at a depth of 1 cm. a well circumscribed mass was encountered. It appeared to be encapsulated. This mass was separated from the spinal cord with little trouble. It was about the size of a little finger, and had a smooth glistening capsule. It was tilted up from its base at the upper pole and seemed to be attached along its lower third to several large blood vessels. The capsule ruptured at this point and a pedicle about 1 cm. in diameter of yellowish "chicken clot" material extruded. A small amount of freshly clotted blood was present at the lower pole of this extruded mass. At this point profuse bleeding occurred from the center of the mass which appeared to be the calcified wall of an aneurysm. This wall was largely removed but profuse bleeding requiring muscle stamps and packing prevented further dissection. The dura mater was then closed, and the muscles approximated. The patient was in fair condition

at the conclusion of the procedure.

The specimen removed was a mass of homogeneous pink-staining tissue with a few elongated fibroblasts and occasional granular material, presumably early calcification. It was concluded that this material represented hyalinized connective tissue in the wall of an aneurysm.

The patient had a rather stormy convalescence requiring Monro tidal drainage. His urine gradually cleared and his anemia was treated by repeated blood transfusions. On January 21 he had a series of six convulsive seizures before the attacks could be controlled by phenobarbital medication. His blood pressure became elevated on January 26, being 174/138. His urine contained ten white and red blood corpuscles and had a moderate amount of albumin. He had another convulsion on the following day. The nonprotein nitrogen of the blood never exceeded 53.3 mg. per 100 cc. The patient continued to have convulsions about twice a week.

On February 12, the Monro tidal drainage was discontinued. His condition gradually improved. On March 31 he complained of blurring of vision and a macular retinitis was found. On April 18 his visual acuity was improving. He could move the right lower extremity, but dorsal flexion of the foot was very weak. The left hip and knee could be moved feebly. Both lower extremities were quite spastic and atrophic. The knee and ankle jerks were absent on both sides and the left plantar reflex was extensor. Sensory disturbances consisted of only a mild hypesthesia in the perianal region and above both ankles. The patient had a neurogenic bladder. He was discharged to his home and has been seen on two subsequent occasions.

On May 18, 1942, he was improving, had no pain, but was still incontinent. He was able to elevate both legs but had a marked analgesia of the left foot. Both knee jerks were active and plantar reflexes were flexor. He was unable to stand. He returned again on November 13, 1942, still having a great deal of difficulty with micturition. He was able to stand up with the aid of crutches, dressed himself, got into bed by himself. He stated that occasionally he felt an urge to urinate. The knee jerks were present on both sides but more active on the right. The right ankle jerk was hyperactive; the left ankle jerk sluggish.

Comment. Vascular anomalies of the spinal cord in children are rare and in those cases reported[3,4,7,8,18] no mention has been made of dilatation of the spinal canal. In retrospect it seems that an angiomatous neoplasm of the spinal cord should have been suspected in this case. The sudden onset with abdominal pain and the presence of xanthochromic spinal fluid on spinal puncture would suggest such a diagnosis for these are typical of a vascular anomaly of the spinal cord.[7] As in the case of Robertson[18] it seems likely that the severe pain initiating the symptoms in this patient was the result of hemorrhage into the spinal cord.

DISCUSSION

The enlargement of the spinal canal in these cases presumably occurred early in the development of the individual, probably at the time the ossification centers were laid down, since except for the last case there was no evidence of spinal cord compression either by lumbar puncture or at the time of operation. In fact, in the first two cases of myelodysplasia there was much more room in the subarachnoid space than usual. If this assumption is correct, it must then be assumed that the segments of the spinal cord did not change their position relative to the vertebral column as normally occurs. At three months of intrauterine life the spinal cord segments correspond anatomically to the vertebral segments, but normally at birth the lower end of the spinal cord lies at the third lumbar vertebra, and in adult life at the first lumbar vertebra. In the lumbar region of the spinal cord, where most of the myelodysplasias occur, this lack of upward migration will cause considerable tension on the spinal cord and it probably is the main reason for symptoms developing early in adult life.

Although the enlargement of the spinal canal has been noted in some autopsy descriptions of diastematomyelia, it has not been recognized generally by clinical neurologists or roentgenologists. In discussing

the roentgenologic aspects of spina bifida occulta, Dittrich[9] does not refer to dilatation of the vertebral canal. In Elsberg and Dyke's paper[10] on enlargement of the spinal canal no mention is made of myelodysplasia as a cause of such dilatation, nor do Pancoast, Pendergrass and Schaeffer[17] refer to it. It might be assumed that the dilatation of the canal associated with congenital anomalies could be distinguished from that due to intraspinal neoplasm by the appearance of the pedicles—the enlargement in the first case being due to a congenital maldevelopment, the pedicles might not be so eroded or thinned as in the second instance where local pressure causes the dilatation. However, such a differentiation does not seem to be possible for the pedicles are thinned in the present series of cases.

SUMMARY

Three cases of myelodysplasia are presented associated with a dilatation of the spinal canal at the site of the anomaly. A case of vascular abnormality—angioma and aneurysm—of the spinal cord accompanied by enlargement of the spinal canal at the site of the lesion is also reported.

The roentgenographic and pathological manifestations of myelodysplasia are discussed.

Although neoplastic conditions cause most instances of roentgenographically demonstrable enlargement of the spinal canal, congenital abnormalities of the spinal cord should be considered as etiological factors.

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CLINICAL AND ROENTGEN MANIFESTATIONS OF DISSECTING ANEURYSM OF THE AORTA*

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INTRODUCTION

DISSECTING aneurysm of the aorta has been known to clinicians and pathologists for many years and the literature contains frequent references to this most interesting condition. However, the number of cases diagnosed correctly during life still remains extremely small. While relatively uncommon, this lesion is nevertheless of great clinical significance. In many instances, the dissection of the aorta develops with dramatic suddenness and is manifested by excruciating pain, collapse, and sudden death. The symptoms may closely simulate coronary disease, embolism or rupture of the heart, and errors in diagnosis are therefore very common. The clinical signs are often misleading and confusing. Since prompt and accurate diagnosis is of the utmost importance from the standpoint of both therapy and prognosis, an attempt will be made in this brief review of the subject to emphasize the important clinical and roentgen manifestations which are of assistance in establishing the diagnosis.

ETIOLOGY

The incidence of dissecting aneurysm of the aorta varies widely in different series of reported cases. In hospital and private practice, the number is doubtless smaller than autopsy statistics indicate. This is owing to the fact that the lesion frequently occurs in persons who have had no previous complaints and death occurred suddenly. Hence, the studies in this condition are more often carried out by the coroner and medical examiner than by the clinician. According to Holland and Bayley,[13] the number of cases of dissecting aneurysm of the aorta is 1 in every 381 necropsies (0.3 per cent). The figures from the San Francisco Coroner's Office for the five year period from 1933 to 1937[13] showed 60 instances of dissecting aneurysm of the aorta in a total of 8,438 autopsies (0.7 per cent). In this series, there were over twice as many cases of ruptured heart, which presented an incidence of 1.5 per cent. At the Boston City Hospital in the five year period from 1939 to 1943 inclusive, there were 4,240 necropsies, of which 15 revealed dissecting aneurysm of the aorta, an incidence of 1 in 283 (0.4 per cent). During this same interval there occurred 11 instances of ruptured heart (0.3 per cent) and 140 cases of coronary occlusion (3 per cent).

The condition is more common in men than in women, the ratio being about 3 to 1. The age distribution varies widely and, although the lesion is most common in middle and late adult life, cases have been reported at all periods from the teens to the ages of eighty and ninety. Trauma and exertion do not appear to play an important part. Occupation similarly does not enter as an etiologic agent. Syphilis is absent in the majority of cases, although a dissecting aneurysm may occur independently in the presence of a luetic aneurysm. Hypertension and atherosclerosis are very important etiologic factors and are found in a large proportion of the cases.

PATHOGENESIS

A dissecting aneurysm is the lesion produced by penetration of the circulating blood between the layers of the wall of a blood vessel. It is not a true aneurysm in that the vessel is not dilated. There is still considerable disagreement as to the exact pathogenesis of dissecting aneurysm of the aorta. Some observers feel that arteriosclerosis is not a predisposing factor, but rather that the initial rupture of the intima which permits the blood to dissect between

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the layers of the blood vessel wall is produced by overstretching of the aortic wall due to the forcible action of a hypertrophied, compensated left ventricle. The great majority of pathologists are of the opinion, however, that the lining and medial wall of the aorta are diseased and weakened by localized destruction and the subsequent processes of repair. This area of repair is weaker than the vascularized fibrous tissues seen, for example, in lues. It is also generally assumed that there is an increase in the blood pressure and usually a tear in the intima. This latter manifestation may not always be demonstrable, however.

The dissection most commonly appears to have its beginning in small, localized areas of atheromatous degeneration. The lamina of the media is split by pressure from above, although as stated previously a tear of the lining cannot always be found. The blood then dissects from the point of rupture and the separation of the coats of the aorta and consequent extravasation of blood between the coats may involve a portion of the circumference or the entire lumen of the vessel. There may be an external rupture or a rupture into the lumen with subsequent formation of two tubes one inside the other, if the patient survives. The lesion may involve the ascending aorta, the arch, descending thoracic portion, the abdominal aorta or any combination of these. Extension of the lesion along the walls of the branches of the aorta is also frequently seen. The dissection is usually in the direction of the blood flow, but at times it also extends toward the heart.

Atherosclerosis is found in the great majority of cases of dissecting aneurysm and in many instances is far advanced and generalized. Syphilis is present in only a small proportion of the cases, however. It appears that syphilis uncomplicated by arteriosclerosis fails to provide the necessary background for the development of dissection of blood between the coats of the aortic wall. In fact, many observers are of the opinion that a granulomatous process such as lues fuses the lamellae more closely together and thereby actually lessens the tendency to dissection.

The arteries originating from the aorta may be affected in various ways by the development of the dissection. There may be complete separation of the artery at its point of origin; or the vessel may be obstructed partially or completely by hematoma formation. The periarterial sympathetic plexus may be damaged with the development of a spontaneous periarterial sympathectomy. Rupture into the mediastinum, the pericardium, or the pleural cavities is common and usually results fatally. Coarctation of the aorta and aortic hypoplasia predispose to dissection. Patients with disease of the vasa vasorum are also apt to develop this lesion.

SYMPTOMS AND SIGNS

In some instances, the onset is so sudden and the course so rapid that clinical diagnosis of dissecting aneurysm is extremely difficult or entirely impossible. The difficulties in arriving at a diagnosis are enhanced by the fact that death frequently supervenes before adequate studies can be carried out. However, as the manifestations are more clearly understood, a definite syndrome is becoming established and the number of cases correctly diagnosed during life is increasing greatly, particularly in recent years. The possibility of dissecting aneurysm must be carefully considered in the differential diagnosis in all patients presenting excruciating chest pain and collapse. As with many other rare and unusual lesions, the diagnosis will be made with ever increasing frequency if the condition is constantly borne in mind.

In the typical acute case, there is a sudden onset of severe, excruciating pain which may be described variously as tearing, crushing or agonizing and is accompanied by a sense of impending death. The intensity reaches its maximum almost immediately and may last for only a few moments or persist for hours to several days. In some instances, the pain is moderate or slight and rarely is entirely absent. The pain may

be located in the chest, abdomen, neck, head, back or lower extremities and usually extends progressively or irregularly from one area to another. The arms are less commonly involved. If radiation to the upper extremities occurs, it is more apt to involve the outer surfaces. As the dissection extends along the aorta, the pain usually spreads rapidly. Hence, radiation in rapid succession from the thorax to the abdomen, thighs and extremities should at once call to the observer's mind the possibility of this lesion. The pain may persist undiminished for a considerable period or become intermittent, lessen or disappear completely. The site of the pain and its radiation vary with the location of the lesion and are important in indicating the portion of the aorta involved. The progress of the dissection may be followed by the varying positions of the symptoms. The pain is usually not precipitated by exertion. The patient is, in most instances, in apparent good health prior to the onset.

Collapse and syncope with shock are frequently severe, although in some cases these manifestations are moderate or slight. There is in many patients, a long history of hypertension. The blood pressure may be maintained at a high level for a considerable period during the stage of dissection, or may drop to a very low level. The blood pressure may be unequal in the two arms and in the legs. The heart is usually enlarged and the rate is rapid. A diastolic murmur is apt to appear in the aortic area. The common signs of aortic regurgitation are absent, however. The peripheral pulses may be altered. The pulse tension and volume of the aorta are diminished because of the partial obstruction of its main branches by the dissection and hematoma formation.

Nausea and vomiting are common. Fever and leukocytosis of a moderately high degree are present in a large majority of cases. The icteric index is elevated and may remain so for days or weeks after the onset. Numbness, coldness, and weakness of the extremities are frequent. Paresthesia and hyperesthesia are less often seen. If the renal vessels are involved, there is hematuria, anuria and uremia. Bleeding into the mediastinum and pericardium may result in cardiac tamponage with marked distention of the cervical veins and an elevated venous pressure.

Neurologic manifestations indicate involvement of the vessels of the spinal cord. Paresis and paralysis are commonly seen and may be variable and inconstant, wandering paralyses being a frequent occurrence in patients with dissecting aneurysm. Permanent paralysis may occur. Urinary retention develops due to sphincteric involvement. Obstructive signs are seen in the peripheral circulation if there is involvement of the arteries arising from the aorta. Convulsions, coma, and hemiplegia may result if the cerebral circulation is interfered with.

The electrocardiographic findings are not, as a rule, characteristic and may actually be misleading. Thus, if the dissection is in the region of the root of the aorta with involvement of the first portion of the coronary artery, acute myocardial infarction may result with consequent changes in the electrocardiogram which cause confusion in the diagnosis.

In the acute case, death may occur at the very onset of the attack or supervene after a variable interval. The average duration of life is approximately two to three weeks. In many instances, the dissection becomes arrested and appears to heal. Most of these patients succumb subsequently, the cause of death being either the development of a new dissection or intractable heart disease. The usual length of life in the old or healed cases is two to three years after the original dissection.

DIFFERENTIAL DIAGNOSIS

Coronary thrombosis is the condition usually confused with dissecting aneurysm of the aorta. The previous history in coronary disease is frequently helpful. In coronary thrombosis, there is absence of the peripheral vascular occlusions and paralyses which may be so prominent in dissecting

aneurysm of the aorta. Syncope at the onset of dissection is an important point in the differential diagnosis.

Embolism and thrombosis of the peripheral arteries and the iliacs may be confused with dissecting aneurysm. In the latter condition, the pain is usually thoracic or abdominal at the onset. Syphilitic aneurysm with rupture may be impossible to distinguish from dissecting aneurysm, especially since the two conditions may exist simultaneously in the same patient. Similarly, rupture of the heart may give a clinical picture closely simulating dissecting aneurysm of the aorta.

TREATMENT

Therapy is entirely expectant and symptomatic. Rest and sedation are of the utmost importance. Correct diagnosis is extremely important to avoid the institution of therapeutic measures which may serve to increase the damage.

ROENTGEN FINDINGS

The roentgen picture, while not clear cut or pathognomonic in all cases, may be of assistance in establishing the diagnosis and in following the progress of the case. The changes vary with the location of the lesion, the extent of the extravasation, and the stage of the process at the time the patient is seen. The most significant finding is widening of the supracardiac area. The increase in width may be to the right, the left or both. It presents a localized bulge if the extravasation is circumscribed or sacculated and a generalized widening if the extravasation is diffuse and widespread. The aortic pulsations are commonly diminished, although in some instances the movements of the aorta are normal or increased. If there is extensive accumulation of blood in the mediastinal tissues, the aortic pulsations are absent. Cardiac hypertrophy is present in many cases and is frequently marked, with vigorous pulsations. This is especially true in long-standing or arrested cases. The enlargement of the heart is in the region of the ventricles. During the acute stage, the cardiac pulsations may be diminished.

The trachea and esophagus are usually displaced. The displacement is to the right or left depending on the site of the dissection. If the lesion involves the large branches of the aorta, there is an increase in the width of the shadow of the innominate artery with a band of increased density in the paravertebral portion of the upper lung field. With extravasation of blood into the pericardium, the findings are those of pericardial effusion. Similarly, rupture into the pleural space produces the usual roentgen manifestation of fluid. Effusion occurs more frequently on the left, but may involve the right side or be bilateral.

In the diagnosis of dissecting aneurysm of the aorta, it is particularly important that the roentgen studies be carried out in close cooperation with the clinician. Doubtless the reason that the diagnosis has been made so seldom in the past is that the possibility of this lesion has not been borne in mind. As it is considered more frequently in the differential diagnosis, dissecting aneurysm of the aorta will be diagnosed with increasing certainty. Since the prognosis is extremely grave in this condition, the importance of accurate diagnosis is obvious. A sudden increase in the width of the aortic shadow roentgenologically is diagnostic of a dissecting aneurysm; similarly, a marked widening of the innominate shadow is very significant. Disappearance of the aortic pulsations indicates extravasation into the pericardium. These manifestations may require careful roentgenoscopic and roentgenographic study and in the acutely ill cases can be performed only with extreme caution and care. Laminagraphic studies of the aorta may be of value in localizing the portion of the aorta involved and delimiting the extent of the process.

CASE REPORTS

CASE 1. J. A., male, white, aged sixty-five. Four months previously, he had been hospitalized with a diagnosis of arteriosclerotic heart disease and cardiac hypertrophy. For several weeks, he had had marked dyspnea on exertion, orthopnea, and mild epigastric pain which was aggravated by food. On the day of the present

admission, he was suddenly discovered by his family in a state of deep stupor.

Physical examination revealed a well developed and nourished male with moist skin and mild stiffness of the neck. The heart was enlarged to the left; there were no murmurs and the rhythm was regular. A_2 was greater than P_2. There were slight dullness and râles at the right base. There was weakness of the right leg and the right knee jerk was less than the left. Both ankle jerks were absent. The Babinski sign was positive on the right. The blood pressure on admission to the hospital was 160/110, whereas previously it had been 240/135. Temperature was 97.8° F., pulse 80, respirations 22. The urine showed a very slight trace of albumin. The red blood cell count was 3,810,000; white cell count 9,600 and the hemoglobin 71 per cent. The Hinton test was negative. Non-protein nitrogen was 43. The electrocardiogram showed left ventricular predominance with ventricular premature beats in lead II and auricular premature beats in leads II and III.

The patient was digitalized and fluids were restricted. The temperature was slightly elevated, pulse continued at about 100, and the respirations varied from 22 to 32. After four days in the hospital, he was conscious and rational and the weakness of the leg disappeared. His blood pressure rose to 218/130. Roentgen examination revealed an increase in the width of the aorta bilaterally and a marked generalized increase in the size of the heart shadow. There were congestive changes in both lung fields and fluid at the right base. On the eleventh day of his stay in the hospital, he suddenly became markedly dyspneic, collapsed and died.

At autopsy, there was found a dissecting aneurysm of the aorta, with rupture into the pericardium; fibrinous pericarditis; hypertensive heart disease; and atherosclerosis of the aorta. The pericardial sac was distended and contained 1,500 cc. of freshly clotted blood. There was fibrinous pericarditis. The heart was markedly hypertrophied and weighed 800 grams. The myocardium was twice normal in thickness. The intima of the aorta presented numerous golden-yellow plaques scattered over its surface; there was no thickening of the aortic wall. About 2 cm. above the base of the left aortic cusp, there was an irregular, transverse laceration of the intima of the aorta. This area was about 2 cm. in length and extended through the intima and media to connect with a cavity filled with freshly coagulated blood lying within the wall of the aorta. The cavity extended downward in the aortic wall to the origin of the aorta and a sinus communicated directly with the pericardium.

Case II. C. W., male, colored, married, aged forty-nine. He was well until three years previously, when he began to have dyspnea occasionally while engaged in his work of carrying baggage at a railroad station. There was no pain in the chest, orthopnea or edema. His physician told him that he had high blood pressure and restricted his activities. The day prior to admission, there was a very sudden onset of extremely severe pain in the left anterior chest, radiating to the left upper quadrant and to the left lower quadrant of the abdomen. He felt extremely weak and sweated very profusely. A hypodermic injection produced relief for a few hours, after which the pain recurred in the left lower abdomen.

On admission, there was dullness and suppression of the breath sounds over the left lower chest. The heart was enlarged to the left and there was a harsh systolic murmur over the precordium; the sounds were of fair quality and the rate was 120. The blood pressure in the right arm was 112/64 and in the left 128/74. The temperature was 98.6° F., pulse 120, respirations 24. The red blood cell count was 5,000,000; white cell count 15,000, and hemoglobin 85 per cent. The urine contained a trace of albumin and many white blood cells. Roentgen study of the chest revealed homogeneous density over the entire left lung field, indicating a large amount of fluid in the left pleural space. He survived three days and died very suddenly.

At autopsy, there was a dissecting aneurysm of the thoracic and abdominal portions of the aorta with rupture into the mediastinum and left pleural cavity. The arch of the aorta presented an oblique rent about 3 cm. in length in the intima along its superior aspect. Opposite this, there was a tear in the adventitia which was separated from the media by a large clot. The dissection extended downward to a point about 6 cm. below the level of the diaphragm. The heart weighed 550 grams and was fairly normal. There was 150 cc. of blood in the pericardial cavity. There was marked atherosclerosis of the aorta.

Case III. A. C., female, white, aged seventy-three. For several years, the patient had shown mental changes with emotional instability, irritability, and loss of memory. One year ago, she had had a slight shock. She was a Christian

Scientist and an accurate history could not be obtained as she was unwilling to discuss her illness. One day prior to admission, she fell while walking in the street and could not rise. When seen in the hospital, she was unable to use her right arm or leg and her speech was so thick as to be practically unintelligible. There was weakness of the left side of the face and a slight droop of the left side of the mouth. The tongue protruded to the left. The heart was enlarged slightly to the left; the sounds were regular and of good quality and the rate and rhythm were normal. There was a systolic murmur at the apex. The blood pressure was 180/100. The right leg was spastic. The red blood cell count was 4,600,000; the white cell count 6,300 to 11,400 and the hemoglobin 89 per cent. The Hinton test was negative. Electrocardiographic studies showed left ventricular predominance and partial heart block. Roentgen examination of the chest showed that the aortic shadow was markedly widened and there was extensive calcification in the region of the arch of the aorta. The patient's condition remained unchanged for about two months, after which time she developed bronchopneumonia and died.

Autopsy showed a healed dissecting aneurysm of the aorta and right common iliac artery. There was a large opening in the aorta in the region of the beginning of the arch; the lumen was divided into two portions and the entire aorta from this point continued as a double vessel with two lumens of nearly equal size. The entire aorta was calcified and sclerotic. The heart weighed 300 grams and was not remarkable except for marked sclerosis and calcification of the coronary arteries.

CASE IV. J. D., male, white, aged forty-two. About three years ago, the patient was hit by an automobile and rendered unconscious. While at the hospital at that time, he learned that his blood pressure was "210." Since the accident, he had had dizziness, poor vision and soreness behind the left ear. During the past year, he had had frequency and nocturia. Twelve hours prior to the present admission, he was suddenly seized with a very severe pain in the anterior portion of the upper chest. The pain was bilateral and did not radiate. There was profuse sweating and alternating feeling of heat and cold. There was no dyspnea. He vomited several times and had a severe, dry cough. On physical examination, the heart was enlarged to the left and the impulse was heaving; the sounds were of fair quality with A_2 greater than P_2. There was a friction rub at the left second interspace anteriorly. The red blood cell count was 4,250,000; white cell count 12,750, and the hemoglobin 87 per cent. The Hinton test was negative; non-protein nitrogen 40; temperature 98° F.; pulse 90; respirations 20. The blood pressure was 170/110. The electrocardiogram showed left ventricular predominance and right bundle branch block. Roentgen examination revealed marked widening of the supracardiac area, particularly in the region of the arch and descending aorta. The heart was enlarged to the left in the region of the ventricle. During his stay in the hospital, he complained of precordial pain and was partially confused mentally. On the sixteenth day, he suddenly complained of severe chest pain and a sense of impending death. Within a few moments of the onset, he died.

At autopsy, there was 2,000 cc. of clotted blood and fluid in the left pleural cavity, 300 cc. of fluid blood in the right pleural space and 500 cc. of amber fluid in the pericardial sac. The heart weighed 640 grams; the epicardium and endocardium were normal. There were a few atheromatous plaques on the valves. At the posterior aspect of the arch of the aorta, there was a ragged, transverse tear in the intima measuring 1.5 cm. in length. The intima was separated throughout the entire circumference of the aorta, with the exception of a very narrow area posteriorly, from the level of the subclavian artery to the proximal portions of the internal iliac arteries. The cavity thus formed contained a small amount of clotted blood at its extreme lower end. Two fresh ruptures of the aorta, each measuring about 2 cm. in length, were present in the superior aspect of the aorta in the region of the junction of the arch and the descending portion. In the intima, there were many yellow plaques with calcification. The atherosclerotic changes were most marked in the abdominal aorta.

CASE V. F. D. L. Q., male, Filipino, aged forty. One year ago, he was ill with a bleeding peptic ulcer and hypertension. His blood pressure at that time was 168/118. Two weeks ago, a physical examination (pre-employment) revealed "high blood pressure." Two nights ago, at 1:30 A.M., he developed persistent orthopnea and discomfort in the chest. Early the following afternoon, he suddenly felt a sharp, burning pain in the epigastrium; the pain was boring and burning and did not radiate. After sedation,

he was sent to the hospital. On admission, the skin was dry and cyanotic. The heart was enlarged to the left, regular, rate 130, with weak sounds and no murmurs. P_2 was greater than A_2. The blood pressure was 100/55 in the right arm and 100/60 in the left. The red blood cell count was 6,000,000; the white cell count 27,600, and the hemoglobin 95 per cent. Non-protein nitrogen was 46. Electrocardiogram showed left ventricular predominance which was interpreted as "possibly coronary thrombosis, but not characteristic." Roentgen studies showed the aorta moderately widened and the heart enlarged to the left. On the second day in the hospital, he suddenly went into shock and died.

Postmortem examination revealed a dissecting aneurysm of the aorta, hemopericardium, aortic atherosclerosis, and cardiac hypertrophy with an infarct of the left ventricle. The pericardial sac contained 200 cc. of clotted blood. The heart weighed 500 grams and there was narrowing of the coronary arteries with numerous atheromatous plaques in the intima. In the aorta, at a point about 1 cm. above the aortic wall, there was a sharp rupture of the intima and a portion of the media. This area measured 4 cm. in length. A smaller slit measuring 1 cm. in length was also present in this region. The wall of the aorta was dissected from its point of origin to the mouth of the celiac artery. There was hemorrhagic extravasation into the adventitial tissues at the root of the pulmonary artery and the aorta. The tissue layers in this region were separated and contained small clots of blood. There were many small atheromatous deposits scattered throughout the entire aorta.

Case vi. W. R., male, white, aged seventy-five. This patient was admitted to the hospital in pain and moaning constantly. He was unable to give any details as to his history and was unaccompanied by anyone who knew the details of his illness. He was extremely restless and appeared very seriously ill. The heart was not enlarged. A rough systolic murmur was heard over the entire precordium. The blood pressure was 122/58 in the left and 104/52 in the right arm. Temperature was 101° F.; pulse 118; respirations 26. The urine contained a slight trace of albumin and many white blood cells. The white blood cell count was 21,000 and the hemoglobin 88 per cent. The Hinton test was negative. Non-protein nitrogen was 33. Roentgen examination revealed marked widening of the aortic shadow and enlargement of the heart outline. He became progressively worse during his hospital stay and died on the fifth day.

Postmortem examination showed an extensive dissection of the aorta involving over one-third of its circumference and practically its entire length. There was also dissection of both common and external iliac arteries, the innominate, the left common carotid, and the left subclavian arteries for a distance of about 3 cm. About 500 cc. of blood was found in the pericardial cavity. The heart weighed 520 grams and showed acute bacterial endocarditis and a healed rheumatic lesion.

Case vii. J. A., male, white, aged fifty-five. About six hours prior to admission, while talking quietly with a friend, he suddenly experienced a sharp pain in the chest. The pain was so severe that he was unable to draw a deep breath; it extended directly through to the back, and no relief was obtained by lying down. He "broke into a cold sweat," and vomited. About seven years previously, he recollected having experienced a similar, less severe attack; otherwise, he had been well. At the time of admission, the heart was enlarged to the left, there was a systolic murmur at the apex, and A_2 was accentuated. The blood pressure was 240/120. The red blood cell count was 4,300,000; the white cell count 20,000 and the hemoglobin 85 per cent. There was a large trace of albumin in the urine. The Hinton test was negative and the non-protein nitrogen 55. Electrocardiographic studies showed left ventricular predominance and auricular premature beats in lead II. Roentgen studies of the chest showed an increase in the width of the supracardiac area to the left in the region of the descending aorta. The heart was enlarged to the left. He continued to feel weak. The blood pressure dropped to 135/75 in the right arm and 140/90 in the left. On the fourteenth day of his hospital stay, he collapsed suddenly and died.

At autopsy, the heart showed coronary sclerosis, otherwise was not remarkable; it weighed 450 grams. There was 500 cc. of clear fluid in the pericardium. In the aorta, at the level of the left subclavian artery, there was an incision-like rupture of the intima and the intima was dissected from this point to the bifurcation of the common iliacs. At a point about 6 cm. below the above described intimal break, a second, similar lesion was present with the formation of a false lumen. There was also dissection of the superior mesenteric artery for a considerable distance.

CASE VIII. P. G., male, white, aged sixty-four. Two weeks prior to admission, he became ill with a cold and developed marked orthopnea. There were no chills or rusty sputum. About twenty-four hours before entering the hospital, he was suddenly awakened by a very sharp pain just below the xiphoid. The pain was aggravated by breathing and radiated to the left shoulder. On admission, he was slightly cyanotic. There were moist râles at the left base. The heart was enlarged to the left, the sounds were of poor quality, the rhythm regular and the rate 92. A to and fro murmur was present over the precordium and there was a palpable thrill in the second interspace on the right. The liver was enlarged and there was edema of the legs. The blood pressure rose to 110/80. The red blood cell count was 4,780,000; the white cell count 9,550, and the hemoglobin 78 per cent. The Hinton test was negative, the nonprotein nitrogen 36 and the icteric index 8. Electrocardiographic studies showed left ventricular predominance and Q-T interval was prolonged by 0.02 second. Roentgen examination revealed a widening of the supracardiac area bilaterally, enlargement of the heart shadow both to the right and left, and congestive changes in both lungs. He was digitalized but continued to have substernal pain and cyanosis. On the third day, the blood pressure fell to 70/30. The following day, he died suddenly.

At autopsy, the pericardial cavity was filled with a soft, dark red blood clot which formed a coat 2 cm. in thickness over the heart. There was a large perforation in the right lateral portion of the ascending aorta at a point about 4 cm. above the aortic valve; the perforation measured about 1 by 0.5 cm. and communicated directly with the pericardial cavity. The ascending aorta showed an aneurysmal dilatation, its circumference measuring 11 cm. as compared with 6.5 cm. in the transverse portion. The intima and media presented a large rupture measuring 7 by 2 cm. There was an accumulation of dark red blood in the adventitial tissue and between the adventitia and the media; this extended along the aorta to the midthoracic region. A few calcified plaques were present in the ascending aorta. The lungs showed congestion. The heart weighed 600 grams and the left ventricle was enlarged. There was marked stensosis with calcification and thickening of the aortic valve. The left coronary artery showed yellow, irregular plaques.

CASE IX. M. M., male, white. The patient had been perfectly well. About 9 P.M. on the day of admission, he suddenly experienced a feeling of dizziness which was soon followed by a severe pain in the region of the jaw. He became pale, dyspneic and sweated profusely. Sudden collapse followed and he was removed to the hospital. On admission, he was restless; the skin felt cold and moist. He complained of substernal pain. The heart was enlarged to the left and the sounds were faint; no murmurs were present. The blood pressure was 80/40; temperature 98° F., pulse 62, and respirations 15. The red blood cell count was 4,200,000; white count 16,000; and the hemoglobin 70 per cent. The Hinton test was negative. The urine showed a very slight trace of albumin and a few white blood cells. An electrocardiogram revealed left ventricular predominance and prolonged Q-T interval of 0.02 second. Roentgen studies showed marked, bilateral widening of the supracardiac area, enlargement of the heart shadow and fluid at the right base (Fig. 1). During his stay in the hospital, he complained constantly of pain in the chest. The blood pressure rose to 90/70. On the sixth day, there was a sudden exacerbation of the pain, the patient gasped and suddenly died.

At autopsy, there was 70 cc. of partially clotted blood in the pericardium. The heart was moderately enlarged, weighing 520 grams. In the superolateral surface of the arch of the aorta at a point about 2 cm. beyond the orifice of the left subclavian artery, there was a transverse cleft in the intima which measured 4 by 1 cm. From this point a tract extended along the right lateral margin of the aorta to the level of the orifices of the renal arteries. The intima of the aorta presented a rough, wrinkled appearance and several scattered calcareous deposits. The pathologist's diagnoses were: dissecting aneurysm of the ascending aorta with rupture into the cavity and an old, healed, dissecting aneurysm of the thoracic aorta; hypertensive heart disease; atherosclerosis of the aorta and the coronary arteries.

CASE X. J. M., male, white, aged forty-eight. The patient entered the hospital after collapsing suddenly in the waiting room of a railroad station. He stated that he had had no sleep during the previous thirty-six hours. He was unconscious for fifteen minutes. On admission to the hospital, his complaints were weakness and a burning feeling in the throat. Ten years pre-

viously, his physician had told him that he had hypertensive heart disease and that his blood pressure was 220. However, he had felt well and had been able to continue his work. At the time of entry, he was drowsy and ashen-gray; the skin was cold and clammy. The maximum cardiac impulse was palpable in the fifth left interspace 13 cm. from the mid-sternal line. There were no cardiac murmurs; the heart sounds were of fair quality; there were occasional extrasystoles; the rate was 80. There were râles with diminished resonance at the left base. The red blood cell count was 3,340,000; white cell count 14,100, and hemoglobin 90 per cent. The urine contained moderate amounts of albumin. The electrocardiogram showed abnormal S-T segments and T waves upright in all leads with low origin in leads II, III, and IV. Roentgen examination of the chest revealed cardiac hypertrophy and increase in the width of the supracardiac shadow (Fig. 2). On his second day in the hospital, he developed moderate pain in the chest and became very restless. The veins of the neck became distended. The blood pressure was 60/50 and pulse 100. The following day, the blood pressure rose to 110/80. His condition remained unchanged until the fifth day when he suddenly developed shock and died.

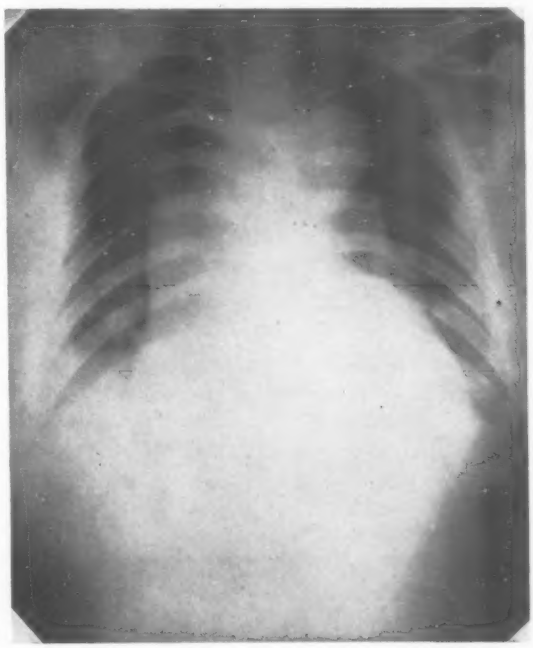

Fig. 1. Case IX. Dissecting aneurysm of the aorta with rupture into the pericardium. The supracardiac area is increased markedly in width bilaterally. To the right, this density is uniform and presents a sharply defined, slightly rounded lateral margin, indicating hemorrhage into the pericardial sac. On the left, the dissection has resulted in an area of density adjacent to the shadow of the descending aorta and the border of the heart. This area is less dense than the shadow of the heart, and the aorta and cardiac border are visible through it. The aortic knob is prominent. The heart is large. There is fluid at the right base.

Fig. 2. Case X. Dissecting aneurysm of the aorta with rupture into the pericardial sac and the right pleural space. There is widening of the supracardiac area both to the right and left. The heart is enlarged. There is fluid at the right base.

At the postmortem examination, there was 500 cc. of sanguineous fluid in the right pleural cavity and 450 cc. of blood in the pericardial cavity. The heart weighed 550 grams and the myocardium was firm and red. The valves appeared normal. The coronary artery was tortuous and dilated. There were several large atheromatous plaques in the ascending portion of the aorta. At a point about 2 cm. above the aortic valve, there was a tear 3.5 cm. in length extending through the intima. There was a dissection of the subadventitial tissue for a short distance from this lesion. A few atheromatous plaques were present in this portion of the aorta, and large atheromatous areas were found in the descending thoracic and abdominal aorta.

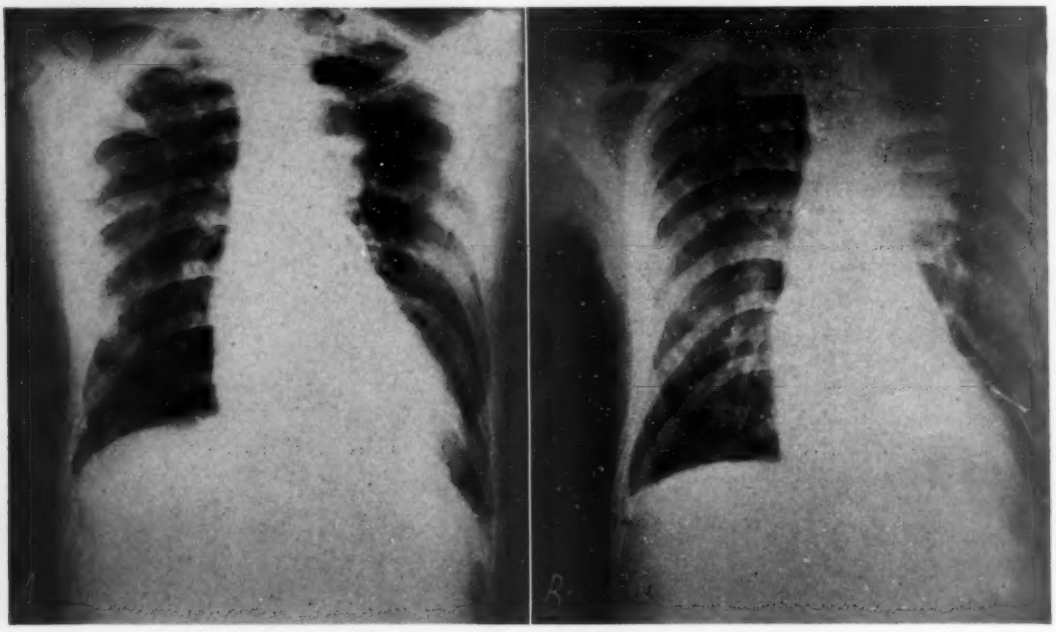

FIG. 3. Case XI. Dissecting aneurysm of the aorta. *A*, there is an area of increased density extending laterally from the shadow of the aortic knob and descending aorta. There is no widening of the supracardiac area on the right side. The heart is enlarged to the left in the region of the ventricle. *B*, same patient five days later. The area of increased density in the left paravertebral region has increased very markedly in size and is more dense than previously. This rapid increase in the width of the aortic shadow is one of the most important findings in dissecting aneurysm.

CASE XI. T. L., male, white, aged thirty-two, taxi driver. Two hours prior to admission, while opening the door of a closet at home, he experienced a sudden, severe pain in the upper mid-back. The pain radiated to the epigastrium and he vomited clear fluid. His face became ashen, his lips blue, and his extremities clammy; he did not collpase, however, and was able to remain standing. Seven years previously, he had been in a sanatorium for sixteen months with tuberculosis and at that time, he was told that he had hypertension and a systolic murmur at the apex of the heart. For the past six months, there had been moderate dyspnea on severe exertion and a sense of pressure in the region of the sternum in cold weather. On admission, he was in acute distress and writhing in bed. The heart was enlarged slightly to the left; the rate was 100 and regular. There was a harsh systolic murmur at the aortic arch and a thrill, transmitted to the neck; there was also a diastolic murmur at the aortic area and a systolic murmur at the apex. The abdomen showed marked spasticity but was not tender. Electrocardiographic findings were as follows: normal sinus rhythm; rate 100; P-R interval 0.16; Q-S interval 0.10; T waves upright in all leads; P waves broad; marked left axis deviation. Roentgen examination of the chest showed an area of increased density in the left mid-chest, adjacent to the shadow of the descending portion of the aorta, and enlargement of the heart to the left in the region of the ventricle (Fig. 3*A*). Re-examination five days later showed the area of density in the left paravertebral region markedly increased in size (Fig. 3*B*). The blood pressure was 180/50 and was equal in the two arms. There were râles at the right base. On the third day, he became irrational and incontinent. Then suddenly he became cyanotic, dyspneic and comatose. A pulsating mass appeared just below the epigastrium. He rallied for a few hours and then died suddenly.

At postmortem examination, 2,000 cc. of fluid blood and clots were present in the left pleural cavity. The pericardial cavity contained 60 cc. of clear yellow fluid. The heart weighed 540 grams and was markedly enlarged. The aorta showed moderate atherosclerosis of the lower thoracic and abdominal portions. Five centi-

meters beyond the orifice of the left subclavian artery, there was a longitudinal tear about 1 cm. in length in the intima of the aorta. The media was dissected from this point down to the iliac arteries. There was dissection of the right renal artery for a distance of 1 cm. with complete occlusion of its lumen.

Case xii. C. D., female, white, aged sixty-eight, widow. Four hours before admission, while climbing a flight of stairs, she experienced a sudden tearing sensation between the shoulders radiating to the epigastrium. The pain lessened but after about one-half hour she developed a complete sensory and motor paralysis below the level of the eighth thoracic vertebra. On admission, she was in shock, cyanotic and pulseless; no blood pressure reading could be obtained. The heart was enlarged to the left and there was substernal dullness with marked widening of the mediastinum. A gallop rhythm was heard over the precordium. The lower margin of the liver was 2 inches below the costal margin. Neurologic studies revealed motor and sensory loss below the level of the eighth dorsal vertebra. There was tenderness over the entire spine. The left knee-jerk was present. The plantar reflexes were weakly extensor. Lumbar puncture showed 90 white blood cells per cubic centimeter, otherwise normal. Electrocardiographic studies revealed a shifting auricular pace maker with bursts of nodal and ventricular tachycardia; the rate was 135; P-R interval 0.06 and 0.20; T_1-T_2 obscured; T_2-T_4 up; left axis; occasional dropped beats. Roentgen examination showed a very marked increase in the width of the supracardiac area bilaterally and enlargement of the heart to the left (Fig. 4). The patient was given clyses of 3,000 cc. of 5 per cent glucose in water. In spite of adequate fluid intake, her output during the first twenty-four hours was only 20 cc. and on the remaining days only about 10 cc. The non-protein nitrogen was 50 on admission and rose to 102 before death. There was no appreciable change in her condition and she died quietly.

At autopsy, a slit 2 cm. in length was present in the intima of the aorta about 5 cm. above the aortic valve. The intima was dissected throughout the entire ascending arch and descending aorta to a point 1 cm. below the origin of the left renal artery. There was dissection of the fifth to twelfth intercostal arteries. The left renal artery had been occluded by the dissection and there was complete infarction of the left kidney. The right kidney and its artery were normal. On the outer surface of the aorta, there was a rupture slightly distal to the aortic valve and within the pericardial cavity. The heart weighed 500 grams, otherwise it was normal. There was 300 cc. of clear yelllow fluid in the right chest and 200 cc. of fresh unclotted blood in the pericardial cavity.

Fig. 4. Case xii. Dissecting aneurysm of the aorta. The supracardiac area is increased markedly in width bilaterally. The heart shadow is enlarged to the left.

SUMMARY

Dissecting aneurysm of the aorta is relatively uncommon; nevertheless this condition is of great clinical significance as it may be confused with coronary disease, embolism or rupture of the heart.

A dissecting aneurysm is produced by penetration of the circulating blood between the layers of the wall of a blood vessel and is usually associated with a tear in the intima of the artery, hypertension, and atherosclerosis. Rupture into the mediastinum, the pleural cavities or the pericardium is common and usually results fatally.

In the acute cases, there is usually sudden onset with severe pain in the chest or

abdomen. The pain is apt to radiate rapidly as the dissection progresses; collapse, shock, and death may occur immediately or be delayed for weeks or months.

The roentgen studies are of value in establishing the diagnosis and in following the progress of the case. The significant roentgen findings are increase in the width of the shadow of the aorta, cardiac hypertrophy, pericardial effusion, and fluid in the pleural space.

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PREPYLORIC DIVERTICULUM OF THE STOMACH DEMONSTRABLE ONLY BY PRESSURE ROENTGENOGRAMS

REPORT OF A CASE*

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DIVERTICULA occur in the stomach much less frequently than in other portions of the gastrointestinal tract.[4] They are usually situated at the fundus in the region of the cardiac orifice. They are rare in other portions of the stomach. Because the diverticulum was located in the lower end of the stomach and because it could be demonstrated only by pressure, this case seems worthy of record.

Opinions concerning the classification of gastric diverticula vary. They have been classified according to etiology as congenital or acquired, according to morphology as true (if they contain all the elements of the gastric wall) or as false (if they do not), and according to mechanism as pulsion or traction types.[4] Rivers, Stevens and Kirklin[4] reported 14 cases proved at operation or autopsy; of 6 of these cases examined roentgenographically only 2 were true diverticula located at the cardiac end, the remaining 4 being secondary to ulcer or neoplasm. It is of interest that 6 of the 14 cases were located in the prepyloric region, 6 at the fundus and 2 midway between the two. All of the 5 gastric diverticula found at autopsy, at the Presbyterian Hospital, were situated in the cardiac end of the stomach. With the exception of the present case, all of the gastric diverticula demonstrated by routine examination, in the Department of Radiology, were located in the same region. Tilger[6] reported a traction diverticulum of the prepyloric region resulting from a gallbladder adhesion.

Gastric diverticula may or may not produce symptoms.[1] Relief of such symptoms has been recorded following surgical treatment.[2,5,7]

The outpouching at the fundus is usually demonstrated with ease and may be differentiated from ulcer, which it may simulate, because it usually retains barium after the stomach is empty. The prepyloric diverticula which have been demonstrated by roentgen methods produced obvious extraluminal barium shadows (Tupper[7]). Sutherland[5] reported a filling defect in the gastric shadow due to pressure of the blood filled diverticular sac on the stomach.

CASE REPORT

A. G. (Unit No. 641925). A sixty year old Austrian tailor came to the Vanderbilt Clinic May 13, 1941, because of a contracture of the right ring finger of three years' duration. The presenting medical problem was whether the patient had Dupuytren's contracture or rheumatoid arthritis. The routine history disclosed that the patient had had one or two bloody stools and recent constipation. This, together with slight epigastric tenderness, led to a roentgen examination of the stomach on May 26, 1941. The roentgenoscopic examination disclosed an extraluminal barium shadow about 1.0 cm. in diameter in the prepyloric region which could be demonstrated only by pressure of the gloved hand and which was completely obliterated by normal peristaltic waves (Fig. 1 and 2). This shadow was reproduced on four pressure roentgenograms (Fig. 3). The long axis of the shadow paralleled the longitudinal rugae. This shadow was considered to be suggestive of an ulcer. A second roentgen examination, July 8, 1941, reproduced this shadow as described above.

Gastroscopy was done by Dr. Armistead C.

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Fig. 1. Diverticulum of the lower end of the stomach. Routine roentgenogram of the stomach in the prone oblique position fails to disclose an extraluminal shadow in the prepyloric region.

Fig. 2. Diverticulum of the lower end of the stomach. Routine roentgenogram of the stomach in the erect position fails to disclose an extraluminal shadow in the prepyloric region.

Crump, July 23, 1941, and revealed what appeared to be a pocket-like depression on the posterior wall in the prepyloric region into which the normal mucosa dipped. Dr. Crump thought that this was not an ulcer and was probably a diverticulum. A second gastroscopy

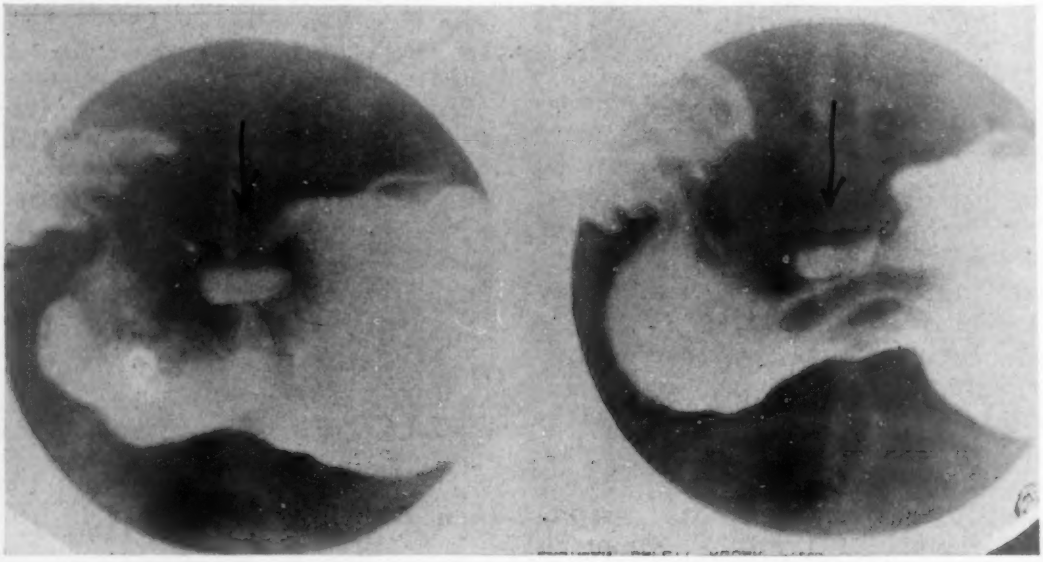

Fig. 3. Diverticulum of the lower end of the stomach. Pressure roentgenograms made May 26, 1941, disclose an oval barium shadow in the prepyloric region suggesting the crater of an ulcer. This shadow was reproduced at a second observation on July 8, 1941.

was done August 20, 1941, and the outpouching was again seen but appeared to be not quite as deep as at the first observation. The gastric mucosa appeared normal.

The patient's arthritic complaints were greatly improved following gold therapy. No digestive disturbances were recorded up to the patient's last visit on January 6, 1942.

COMMENT

This case illustrates the importance of pressure methods of roentgen examination of the stomach, as this diverticulum was in the posterior wall and was too shallow to be demonstrable in any other way. It also emphasizes the helpfulness of gastroscopy with roentgen examination, as the former showed clearly that the mucosa was intact and therefore that no ulcer was present. The fact that normal peristalsis flowed freely through the antrum and effaced the barium shadow should have suggested that the lesion was not an ulcer. An exact classification cannot be made without histological study. It would seem likely that this was a true diverticulum but whether it was congenital pulsion in type cannot be determined.

SUMMARY

An extraluminal barium shadow in the lower end of the stomach, demonstrable only on pressure, was shown by gastroscopy to be a diverticulum.

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MEGA-ESOPHAGUS AS A CAUSE OF MEDIASTINAL WIDENING*

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EXTENSIVE mass surveys of apparently healthy adults to detect unknown cases of tuberculosis have been a major part of the services of the Bureau of Tuberculosis of the Department of Health since 1933.[1] This type of case finding has been greatly extended as a result of the routine chest roentgenograms of the selectees at the Army Induction Center.[2,3,4] Similar mass studies of adult populations are developing throughout the country so that greater and greater samples of the population are being roentgenographed for the first time.

While the detection of tuberculosis has been the prime objective of such studies, they have at the same time revealed a wealth of non-tuberculous conditions demonstrable in the chest roentgenogram that has been of the greatest interest. For, as in incipient tuberculous lesions, the discovery of many of the non-tuberculous abnormalities has resulted in therapy or corrective measures that have benefited the individual.

This report will deal with 11 cases in which the initial roentgenogram suggested the presence of a mega-esophagus subsequently verified by the barium meal. This condition has in the past fallen mainly within the province of the gastroenterologist and internist. When the dilatation becomes sufficiently marked to project beyond the cardiac or sternal border it presents a rather characteristic appearance on the chest roentgenogram. It therefore assumes an important position in the differential diagnosis of mediastinal abnormalities particularly to physicians interested in chest diseases and to those reading survey roentgenograms.

A review of the literature is confusing in that cases appear classified under a variety of terms. The term "cardiospasm" was first introduced by Mikulicz[8] in 1888 who described the dilatation as due to an actual spasm of the cardiac sphincter. Sturtevant[11] reviewed the literature up to 1933 and defined "cardiospasm" as the "common name used for a condition in which, without demonstrable obstructive pathologic change, and usually without pain, food does not pass readily from the esophagus into the stomach, but is held in the esophagus which in the majority of cases, undergoes dilatation, sometimes extreme."

It has been noted by many observers that the marked dilatation seen in simple, so-called non-organic "cardiospasm" is rarely seen in organic stenosis. No benign or malignant stricture causes dilatation even approaching that produced by "cardiospasm." Some authors have predicated vagus nerve involvement by trauma, lead poisoning, infection, etc., as an explanation for the esophageal ectasia. Others have suggested a congenital tendency to idiopathic dilatation, perhaps similar to Hirschsprung's disease. Finally, denying the existence of a sphincter, the possibility was suggested that the diaphragmatic muscle might cause the obstruction, acting as a pinchcock on the cardia. Recently Etzel[5] proposed a chronic vitamin B_1 deficiency as the basic cause for this condition as well as for megacolon and mega-ureter.

In early studies in 1913, A. F. Hurst[6] stated that the obstruction was caused by absence of relaxation of the sphincter which normally occurs when, in the act of swallowing, a peristaltic wave reaches it. He proposed the use of the term achalasia, meaning absence of relaxation of the cardiac sphincter. He felt that achalasia was not purely functional, a theory widely held, but that investigation would prove that it was the result of organic change involving the neuromuscular control of the sphincter.

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Rake[10] later demonstrated round cell infiltration in Auerbach's plexus at the lower end of the esophagus in early cases and degenerative changes resulting in more or less complete disappearance of the ganglion cells in late cases. Several investigators have confirmed this finding, but contradictory opinions are still held.

A very complete and scholarly presentation of the anatomy of the cardiac orifice of the stomach was recently presented by Lendrum.[7] He was unable to demonstrate a true sphincter or increased local musculature or elastic tissue, but he stated that the last inch of the esophagus acted in that capacity. He gave a complete description of the pathologic findings in 13 cases of "cardiospasm." In no case was there organic narrowing of the neck or scarring around it. No thickened sphincter or hypertrophic oblique muscle was found, nor was there sufficient evidence to implicate the diaphragm in causing compression. In 10 of the 13 cases where adequate sections could be obtained, the vagi were normal. In every case, however, there was a striking loss or complete absence of the ganglion cells from the myenteric plexus.

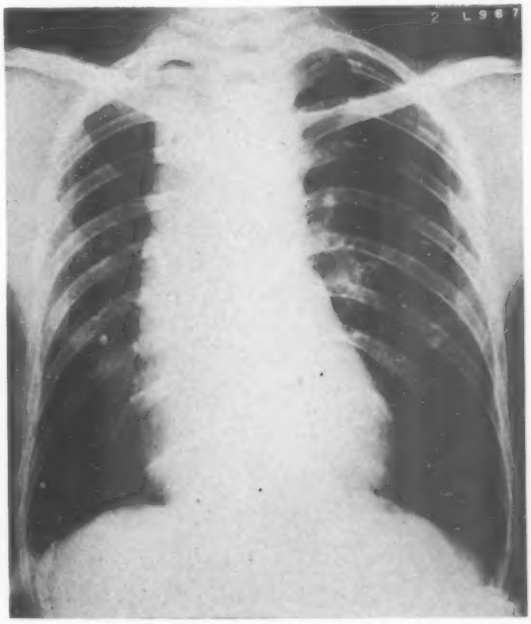

Fig. 1. Case 1.

Fig. 2. Case 1.

REPORT OF CASES

Case 1. M. M., a white male, aged thirty-seven, was rejected by the Armed Forces Induction Station for "cardiospasm." He gave a history of difficulty in swallowing which dated back to 1937 following an attack of pneumonia. There were frequent attacks of vomiting. A diagnosis of "cardiospasm" had been made at a hospital in 1937. Physical examination showed parasternal dullness on the right. The roentgenogram showed a diffuse widening of the mediastinum beginning at the extreme thoracic inlet at D_2 with a curved border proceeding straight down parallel to the sternum and occupying the inner half of the right chest. At the dome of the diaphragm, the density curved to the left obscuring the cardiophrenic angle. There was an air pocket at the extreme upper portion with a mottled stippled appearance in the upper third of the density (Fig. 1). Barium filling showed a narrowing at the cardiac end with a markedly dilated esophagus extending straight down and then assuming a tortuous appearance lying across the dome of the diaphragm (Fig. 2). The esophagus measured 7 cm. from the right border to the mid-point of D_7.

Case 11. M. T., a Negro female, aged thirty-seven, was referred to one of the district clinics

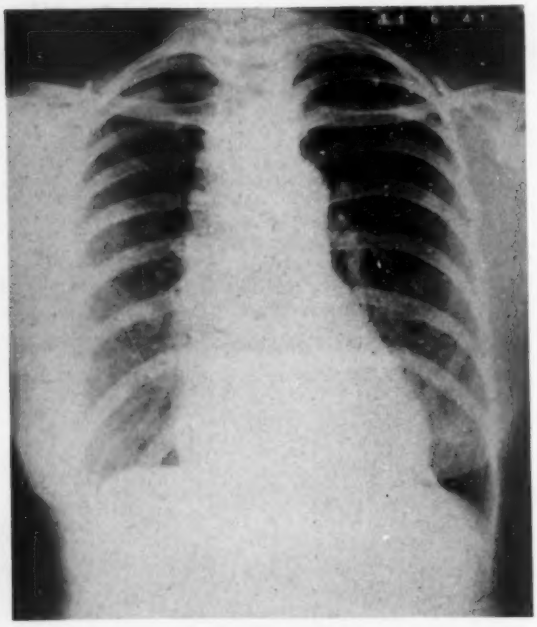

FIG. 3. Case IV.

by her private physician because of cough and expectoration of three weeks' duration. Examination was negative but roentgenoscopy and roentgenographic examination revealed a diffuse mediastinal widening with a density on the right, separate from the heart shadow. She was recalled on the suspicion of an esophageal dilatation. Further questioning revealed a history of some dysphagia, nausea, and occasional postprandial vomiting for the past seven years. The roentgenogram revealed the density to begin at about D_3, without a visible air pocket or fluid level. The density was homogeneous without any stippling and extended straight down reduplicating the cardiac contour and traversing and faintly obscuring the cardiophrenic angle. The density was widest at D_9 and measured 5.5 cm. from the right border to the middle of the vertebra. Barium studies demonstrated the "cardiospasm" and mega-esophagus.

CASE III. S. F., a white male, aged thirty-five, was rejected by the Induction Station for a known "cardiospasm." He had been well up to the age of fourteen when he began to regurgitate food after almost every meal. A diagnosis of "cardiospasm" and mega-esophagus was made, and at the age of twenty-one the anterior vagus nerve was sectioned with only slight relief. Physical examination revealed nothing of note. The roentgenogram revealed a homogeneous density beginning at D_3 and extending downwards reduplicating the cardiac contour and obscuring the cardiophrenic angle. There was a faint suggestion of an air pocket but no fluid level was visualized. The density measured 5 cm. from the middle of D_8 at its widest point. Barium studies showed a high grade "cardiospasm" with a typical mega-esophagus.

CASE IV. L. C., a white female, aged fifty, was referred for examination as a teacher-applicant. There were no complaints or abnormal physical signs but the roentgenogram (Fig. 3) showed a diffuse widening of the mediastinum beginning at D_3 with a soft convexity outwards and downwards. At the upper portion was an air pocket with a questionable fluid line at the level of the clavicles. The upper third of the density had a stippled appearance. The density measured 5 cm. at its greatest width to the middle of D_7. The patient was recalled for further study with the tentative diagnosis of mega-esophagus and gave a history of intestinal upset about ten years previously. Studies done at that time had revealed a dilated esophagus. Barium studies at this clinic (Fig. 4 and 5) showed that the apparently even widening was actually hourglass, the right hilar density bridging the upper and lower convexities. The esophagus was evenly widened to D_{10} and became tortuous beyond that point, lying across the dome of the

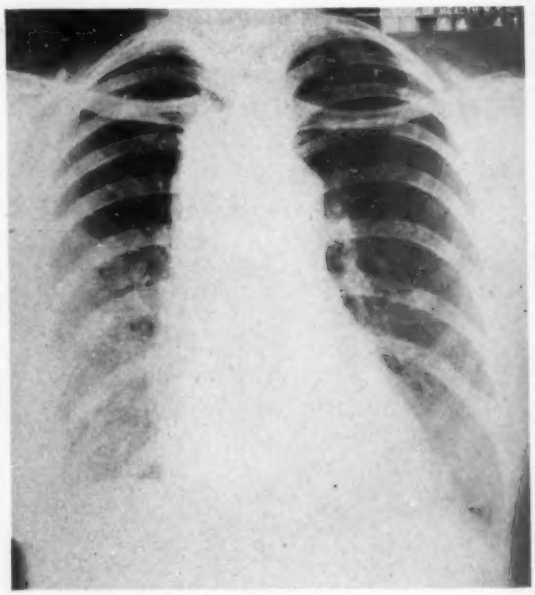

FIG. 4. Case IV.

diaphram. On the right lateral view (Fig. 6) a definite fluid level is noted in a goblet-shaped, stippled area lying between the vertebral column and the trachea which is displaced forwards.

Case v. N. P., a white male, aged thirty-five, was rejected by the Induction Center because of an unusual mediastinal density. He gave a history of "food getting stuck frequently," without vomiting or weight loss for the past five to six years. Physical examination was entirely negative. The roentgenogram showed a

Fig. 5. Case iv.

slight widening of the right upper mediastinum beginning at D_3. Inspection of the $4 \times 10''$ roentgenogram taken at the Induction Center revealed a suspicion of a fluid level at the sternoclavicular junction with an air column above. Maximum width of the density was 4 cm. at the middle of D_6. There was an obscured cardiophrenic angle without reduplication of the cardiac contour. Barium studies revealed a "cardiospasm" with a mild degree of mega-esophagus.

Case vi. M. D., a white male, aged thirty-nine, was rejected by the Induction Center because of the discovery of an unusual mediastinal density. He gave a history of difficulty in eating

Fig. 6. Case iv.

large meals, fullness and the necessity for drinking large quantities of water with his meals. Physical examination was negative. The roentgenogram (Fig. 7) showed a mediastinal widening to the right originating at D_2. There was a

Fig. 7. Case vi.

FIG. 8. Case VI.

suggestion of a fluid level at D_3. The outer density reduplicated the right cardiac contour and crossed the diaphragm obscuring the cardiophrenic angle. The density was widest at D_6, measuring 5.5 cm. to the middle of the vertebral body. No stippling was seen on the posteroanterior view. The lateral projection (Fig. 8) showed a goblet-shaped, stippled density lying between the vertebral column and the trachea which was convex anteriorly. Barium studies revealed a high grade "cardiospasm" with a marked mega-esophagus.

CASE VII. P. A., a white male, aged twenty-two, was first seen in a union survey. He gave a history of having been under observation in a hospital three years previously, was told that he had ulcers of the stomach and was advised to go on a milk and cream diet. No roentgenograms were taken at that time. For the past three or four years he complained of recurrent vomiting immediately after meals, with occasional attacks of left upper abdominal pain and marked belching which usually preceded the vomiting spells. He had lost about 7 pounds in the past year. There had been two occasions in the past year when very black stools were observed. Physical examination was essentially negative. The roentgenogram (Fig. 9) showed a large homogeneous density in the upper half of the right chest extending for a distance of about two-thirds of the transverse diameter of the right chest with a sharp convex outer margin. The border was continuous below with a density reduplicating the cardiac contour and traversing and obscuring the cardiophrenic angle. The widest diameter of the upper rounded portion was 10 cm. to the middle of D_4. Lower down at D_8, the diameter was 7.5 cm. The upper widened portion presented a stippled appearance. A diagnosis was made of mega-esophagus with a diverticulum-like dilatation in the upper portion. Roentgen studies after a barium meal confirmed the diagnosis. With the first swallow of a thick barium mixture the left portion of the upper density filled readily. The right-sided rounded portion of the density filled only after the barium reached the middle third of the esophagus (Fig. 10 and 11).

The patient was hospitalized for further study. The following is a verbatim report of the roentgen findings. "Examination of the alimentary tract by means of a barium meal shows evidence of enormous dilatation of the entire esophagus which in its upper third courses to the right of the spine and produces the shadow of increased density seen on the chest film. A large amount of retained food particles and secretion is present in the esophagus. During the thirty minute observation period none of the barium entered the stomach. The cardiac end is

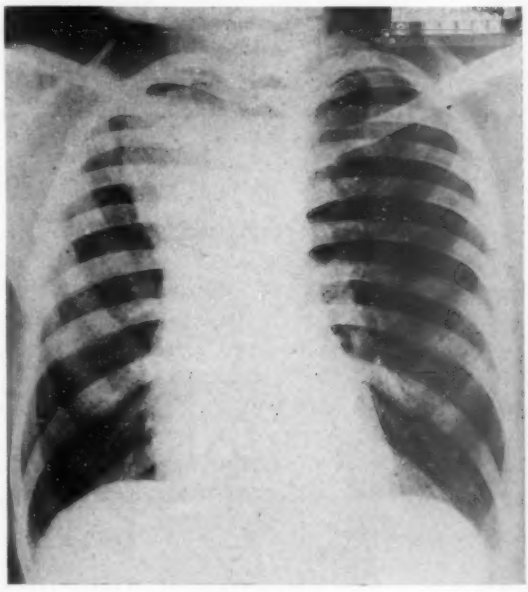

FIG. 9. Case VII.

smooth in contour. No evidence of malignancy can be demonstrated, although this possibility cannot be entirely excluded. Six hours after the ingestion of the barium none of the opaque medium has entered the stomach. Twenty-four hours later barium is seen outlining most of the colon.

"Impression: The findings indicate marked dilatation of the entire esophagus, probably on the basis of a 'cardiospasm.' The obstruction at the cardiac end is of a very high degree."

Case viii. H. A., a white male, aged seventeen, was rejected by the Induction Center for "cardiospasm." Two years previously, while at a hospital for minimal tuberculous lesion, he was informed of this condition. He complained of a choking sensation during his meals for which he has been taking belladonna with slight relief. The roentgenogram showed a slight mediastinal widening beginning at D_4 which followed the cardiac contour and was widest at D_{10}—5 cm. Administration of barium demonstrated a widened esophagus with an hour-glass shape.

Case ix. T. J., a white male, aged twenty-four, had been roentgenographed three years previously and had been told that the findings were negative. He was drafted in 1941 and, following the onset of a respiratory infection, was admitted to the station hospital. Roentgen studies revealed a dilated esophagus. He was given a medical discharge and admitted to the Central Chest Clinic for further study. He had no digestive disturbances but did confess to occasional vomiting which occurred sometimes at night or after eating too quickly. The roentgenogram showed a mediastinal widening beginning at D_2, and becoming progressively more marked up to D_{10} where it was 7 cm. A definite fluid level was noted at D_3 and stippling was present throughout the visible portion of the esophagus. Roentgen studies after a barium meal confirmed the presence of a markedly dilated esophagus.

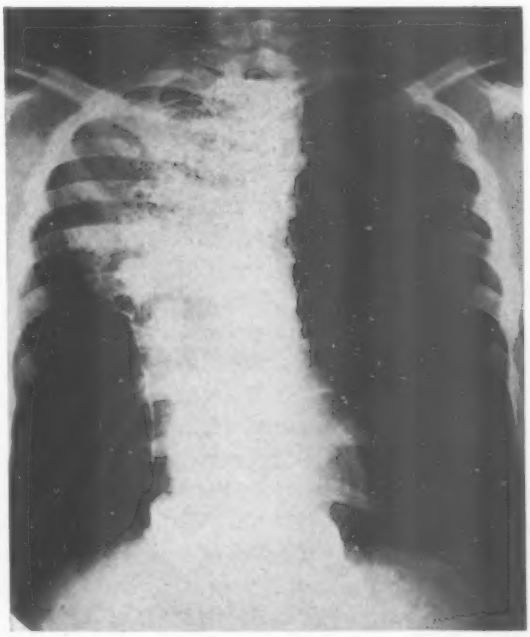

Fig. 10. Case vii.

Fig. 11. Case vii.

Case x. C. J., a Negro male, aged twenty-eight, was referred to one of the district clinics for cough, expectoration, and dyspnea. He gave a history of difficulty in eating for the previous sixteen years and stated that he had to drink water or milk with his meals to get the food down. A diagnosis of widened esophagus was made at a hospital six years before. The initial roentgenogram taken at the district clinic showed a moderate dilatation of the lower mediastinum. On further examination one month later, there was diffuse widening beginning at

D_2 and becoming progressively wider until D_{11}—8.5 cm. at the diaphragmatic level. Barium studies showed a fish-hook type of mega-esophagus with a thin trickle of barium through the cardia.

CASE XI. S. S., a Negro male, aged twenty-eight, was rejected by the Induction Center for "cardiospasm." For the past one and one-half years, he had suffered from cough followed by regurgitation and vomiting immediately after eating. No pain or weight loss had been noted. A diagnosis of "cardiospasm" had been made at a hospital with unsuccessful attempts at dilatation. The roentgenogram showed a diffuse mediastinal widening beginning at D_2 and widest at D_7—8.5 cm. From that point down it became somewhat narrower. A fluid level was clearly seen at D_3 with a suggestion of stippling. On the lateral view, the trachea was convex anteriorly. Roentgen studies after a barium meal showed an enormously dilated esophagus coiled and invaginated in the lower half and appearing at the left of the sternum.

DISCUSSION

There were 11 cases in this series, 9 males and 2 females, ranging in age from seventeen to fifty. It is not possible to draw any conclusions from the age or sex distribution inasmuch as the majority (7) were diagnosed among rejected draftees, all male, and of a selected age group. The others were found as follows: 2 on routine chest roentgenograms taken in the course of a survey, and 2 in a consultation chest clinic referred by their private physician for pulmonary complaints. Among the 11 cases, there were 8 whites and 3 Negroes.

One of the most interesting features in the study of mega-esophagus is the marked disproportion between the relative paucity of the complaints and the good general condition of the patient on the one hand, and the large size of the esophagus on the other. The complaints dated back to from one and one-half years to as long as twenty-one years. There was no definite recollection in most cases of any precipitating cause for the onset of symptoms. The symptoms were varied but referable to the upper digestive tract. In only 1 case was there only a "mild intestinal upset" which had led to the diagnosis. Most cases had difficulty in swallowing, nausea, and vomiting either occasionally or after every meal. Difficulty in eating large meals was a prominent complaint but pain was rarely mentioned. Many authors have stressed the onset of complaints as following some psychic trauma. In Plummer and Vinson's large series,[9] they noted an inverse relationship between psychoneurosis and dilatation of the esophagus. Patients with "cardiospasm" without esophageal dilatation were frequently of psychoneurotic type. Concerning "cardiospasm" with dilatation, they believe that "contrary to general belief, cardiospasm is not a psychoneurosis, and patients suffering from it are usually calm and well balanced with normal nervous systems."

In true mega-esophagus, the variation in the clinical pattern depends upon the ability of the esophageal musculature to overcome the obstruction. This has been compared to cardiac disease in its compensated and decompensated states.[7] In moderate cases, there may be only slight dilatation with thickening of the muscular walls, most particularly the circular muscles. There is also thickening of the circular elastic fibers. If long continued and severe pressure from within continues, other changes may occur. There may be enormous dilatation of the esophagus, with stretching lengthwise and thinning of the muscle. In severe cases, the lower half of the esophagus telescopes upon itself, the muscle fibers are torn and become fibrotic and the elastic fibers thinned. In occasional cases, the mucosa may herniate through the remnant of the muscular wall in the form of a diverticulum (Case VII).

Reference has been made to several diagnostic roentgen features which are here more fully discussed and which will aid in the differential diagnosis of this condition from other causes of mediastinal widening.

1. *Diffuse Widening of the Mediastinum.* The shape of the mega-esophagus depends largely upon the degree and location of the

dilatation. In addition to the diffuse widening, an elongation takes place which may best be described as the coiling on the floor of a slack rope suspended from the ceiling. The widening of the mediastinum is usually seen beginning at the thoracic inlet or just below. In 5 cases, it began at D_2, in 4 cases at D_3, at D_4 in 1 case, and in Case VII an enormous diverticulum was present at the extreme thoracic inlet. The degree of widening is difficult to measure since the greatest visible dilatation takes place to the right of the sternal or cardiac border, and because the left border of the esophagus is lost in the cardiac density. It was not considered practical to measure the esophagus after filling with barium since it was noted that its size, then, bore no constant re'ationship to that in a resting state. With a marked "cardiospasm," the esophagus could dilate to almost double its size to retain the entire barium meal, with a concomitant stretching and thinning of the wall. The largest width of the esophagus on the routine roentgenogram was measured from the right border to the center of the appropriate vertebral body. This varied from 4 cm. at D_6 in Case V to 8.5 cm. at D_7 in Case XI and D_{11} in Case X.

2. *Fluid Level.* A definite level was found in 4 of the 11 cases traversing the tracheal column of air at the level of the clavicles. It depended on recent eating, drinking, and swallowing of air for its presence, and when found was pathognomonic. While present in only 4 cases, it is most important as a diagnostic clue in the absence of marked dilatation sufficient to cause obvious widening of the mediastinal shadow.

3. *Stippling.* In the upper esophagus lying to the right of the sternum, there is occasionally seen a peculiar stippled, marbled, or mottled density. This is due to the contrast created by the mixture of air and food contents. On the right lateral view, this may be demonstrated as a goblet-shaped stippled density lying between the vertebral column and the trachea displaced anteriorly. In 4 of the 11 cases, stippling was a pronounced feature.

4. *Apparent Widening of the Cardiac Contour.* On a routine roentgenogram with normal exposure the right inferior border of the dilated esophagus may give the impression of an enlarged cardiac silhouette on the right and with greater exposure, an apparent reduplication of the cardiac contour is seen. This was present in all but one case which showed the least degree of widening. In addition, in a number of the cases a superimposed density was seen behind the cardiac shadow to the left of the midline. This could best be demonstrated on a roentgenogram with slightly increased penetration and consisted of the redundant lower portion of the esophagus lying on the dome of the diaphragm.

5. *Obliteration of the Cardiophrenic Angle.* In all cases the dilated esophagus, after following the cardiac contour, crosses to the left, the redundant esophagus obscuring the cardiophrenic angle.

In every case, complete barium studies were done to demonstrate the "cardiospasm," stomach, and small bowel. This is very important in order to differentiate between mega-esophagus and congenitally short esophagus with thoracic stomach. The redundant lower esophagus associated with mega-esophagus after filling with barium usually shows incisurae and peristaltic patterns almost indistinguishable from the stomach itself. It is therefore necessary to complete the barium studies to rule out a thoracic stomach.

SUMMARY

1. Mega-esophagus may be diagnosed in the course of roentgen surveys of the chest.

2. Eleven cases have been presented, all of which were diagnosed before roentgen studies with the barium meal.

3. The important diagnostic points were found to be diffuse widening of the mediastinal shadow, fluid level at the level of the clavicles, apparent widening or reduplication of the cardiac contour, stippling of the upper third of the density, and obliteration of the cardiophrenic angle.

4. In suspected cases, complete roentgen

studies with the barium meal should be done including visualization of the stomach and small bowel, to differentiate this condition from congenital anomalies of the esophagus and stomach.

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Vol. 52, No. 6

UNUSUAL TYPE OF INVERSION OF THE STOMACH ASSOCIATED WITH DIAPHRAGMATIC EVENTRATION AND OTHER ANOMALIES

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INVERSION of the stomach, according to Bockus,[3] "is characterized by an orad position of the greater curvature together with extreme left lateral position of the cardia. The duodenal cap is downward . . . ." He goes on to say that "This anomaly may be associated with annoying dyspeptic symptoms and bowel dysfunction."

REPORT OF CASE

Present Illness. A soldier, aged twenty-four, was admitted to the Station Hospital for study complaining of "gas on the stomach," anorexia, occasional vomiting, nervousness, and slight post-prandial left upper quadrant pain which was relieved by belching and lying down after meals. He vomited four or five times a week; the vomitus was free of blood but contained food just previously eaten. There was no constipation or obstipation.

Social History. He has had an unhappy family background, and has taken to excessive smok- and drinking for the past two years. He admits that he has had difficulty in adjusting himself to Army life and was demoted for inefficiency, his symptoms being markedly aggravated directly after this event.

Physical Examination. Upon admission to the hospital, this patient appeared well nourished and not acutely ill. He was both nervous and apprehensive. The examination of the cardiorespiratory system was negative. An old operative scar was present in the right lower quadrant. The abdomen was normal except for a rather firm mass, later shown to be the right kidney, which was felt near the cecum.

Laboratory Findings. Red and white blood cell counts were within normal limits. Urinalysis and serological examination were negative. Free and total gastric acidity was within normal range. Occasional occult blood was noted in the stools.

Roentgen Findings. Roentgenographic and roentgenoscopic examinations of the chest revealed a markedly elevated left dome of the diaphragm, the apex being at the level of the fourth interspace anteriorly, on deep inspiration (Fig. 1). The outline of the diaphragm was smooth and a large gas bubble was noted, which was later shown to be the Magenblase. In the Trendelenburg position, the bubble did not rise above the diaphragm, even on deep abdominal pressure. The left dome of the diaphragm moved approximately 1 inch and its excursions were diminished as compared to the right. No paradoxical movements were noted.

Barium meal examination showed the esophagus to be in the midline; it appeared normal. The cardiac sphincter and cardiac end of the stomach appeared to be at the twelfth dorsal vertebra, while the greater curvature of the stomach was situated at the level of the sixth dorsal vertebra, and fitted closely beneath the elevated left dome of the diaphragm (Fig. 2). The stomach assumed an inverted fish-hook appearance, curving from right to left with the antrum and duodenal cap pointing downward and situated in the left upper quadrant. The

FIG. 1. Marked elevation of the left dome of the diaphragm and large stomach bubble below it.

FIG. 2. Complete inversion and reversal of the stomach with the cardiac end (*C*) situated in the left side of the abdomen at the level of the twelfth dorsal vertebra, the greater curvature margin (*GC*) fitting under the elevated left dome of the diaphragm, and the duodenal cap (*D*) also in the left side at the level of the eleventh dorsal vertebra.

duodenal sweep assumed a normal "C"-shaped curve. With the exception of a few loops of the terminal ileum, almost all of the small bowel appeared in the left side of the abdomen. At six hours (barium water meal used) the stomach was empty and the head of the meal was at the mid-transverse colon. It was noted that the cecum was situated high in the abdomen (Fig. 3) and that the terminal ileum appeared long and dilated.

In the barium enema examination the barium entered the rectum and passed around the cecum without any interference. It was noted that the transverse colon extended diagonally down across the abdomen and the usual well defined hepatic flexure was absent. The tip of the cecum was situated at the level of the crest of the ilium. The colon exhibted a normal degree of mobility throughout. After evacuation a small quantity of barium remained in the colon.

Intravenous Pyelogram. The left kidney appeared normal in size, shape and position; the renal pelvis and calyces were well delineated. In the erect position the left kidney shifted downward approximately 2 inches. Excretion of the opaque medium was excellent. The right kidney was small as compared to the left, and was situated low in the abdomen, the upper pole being at the level of the upper border of the fourth lumbar vertebra with the lower pole extending down to the first sacral segment; only the superior major calyx was well outlined. The right ureter was fairly well delineated and did not appear redundant or looped. In the erect position, the right kidney descended approximately 1½ inches. Excretion of the opaque medium was good (Fig. 4). It can be reasonably assumed, with the information at hand, that the right ureter is short and the low position of the right kidney is not due to ptosis but to arrest in its upward migration to the usual lumbar site.

Final Roentgen Diagnosis. (1) Eventration of the left dome of the diaphragm; (2) complete inversion of the stomach; (3) abnormally rotated stomach, the antrum and duodenal cap being situated in the left upper quadrant; (4) incomplete descent of the cecum; (5) simple iliac ectopia of the right kidney.

COMMENT

It is my opinion and that of others who

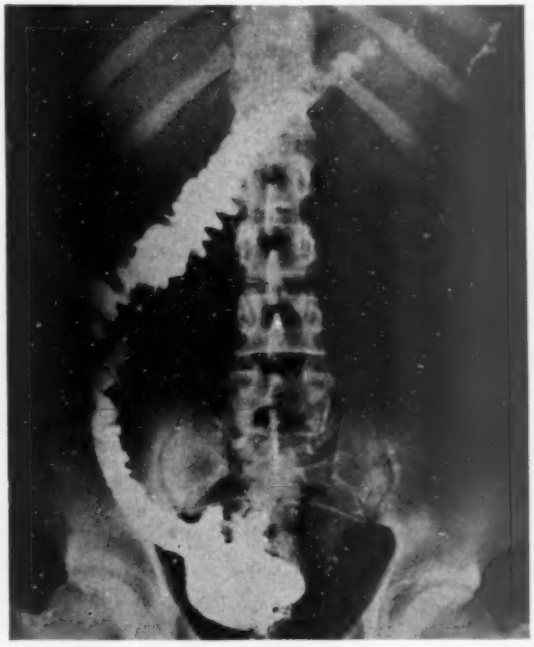

FIG. 3. Six hours after ingestion of the barium and water meal. Illustrates diagonal direction of the transverse colon, lack of hepatic flexure, and high position of the cecum.

have had closer clinical contact with this patient that his complaints were psychogenic in origin rather than due to the many anomalies present. This is borne out by the cases that Ekstrom[5] reported in which she stated, "Neither of the cases had noteworthy symptoms and the discovery of the abnormality was an incidental finding."

A psychiatric study of this patient showed him to be sufficiently maladjusted and mentally unstable as to make him unfit for military service. He was later discharged from the Army.

INCIDENCE

Gross abnormalities of the stomach are rare and reports of inverted or "upside-down" stomachs are exceedingly rare. Bockus makes mention of this condition in his recent text, while nothing is said of this anomaly in the standard treatises on the gastrointestinal tract by Eusterman and Balfour,[6] Barclay,[1] Rehfuss,[10] and others. Feldman[7] states that he saw 2 cases in approximately 15,000 gastrointestinal examinations. Rhinehart and Rhinehart[11] reported 2 cases and Ekstrom,[5] 2 cases. A search through the literature of more than twenty years fails to reveal any others. The most interesting part of the case herein described is that the stomach is not only inverted but reversed, the pylorus and duodenal cap being situated in the left side of the abdomen (Fig. 2), while in the cases already reported the pylorus and duodenal cap are in the right side (Fig. 5).

Fig. 4. Intravenous pyelogram (five minute film). Demonstrates low position and small size of the right kidney. Subsequent pyelograms show the right ureter more clearly.

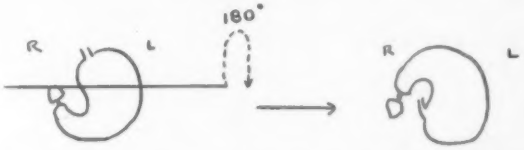

Fig. 5. Diagram illustrating Rhinehart and Rhinehart's conception of the mechanism of gastric inversion of the type that they described.

EMBRYOLOGY

All writers agree that inversion of the stomach is a congenital anomaly and the fact that it is usually associated with other congenital defects adds weight to this contention. There is considerable difference of opinion as to the cause of, and as to the forces that come into play in the production of various normal developmental rotatory phenomena of the gastrointestinal tract, such as the 90 degree rotation of the stomach on its long axis, and the 180 degree counterclockwise rotation of the small bowel around the superior mesenteric artery. One could hardly expect, then, a precise description of the developmental process of this rare abnormality, inverted stomach.

Rhinehart and Rhinehart surmised that this anomaly, of the type they and others described, was the result of rotation of 180 degrees of the stomach around its transverse axis (Fig. 5). I showed the case under consideration to Dr. Corner[4] and his associates at the Carnegie Institute of Embryology. They were rather reluctant to offer

an opinion, since nothing is known as to the mesenteric and ligamentous attachment of the stomach and neighboring viscera. They suggested, though, that a likely explanation was the following: At some time before the duodenum was fixed, when the embryo was between 30 and 35 mm. in size, the stomach was either pushed or pulled up in the left

FIG. 6. Diagram illustrating mechanism of gastric inversion of the type described by the author. See text.

upper quadrant to fit under the elevated left dome of the diaphragm with the cardiac end remaining stationary and acting as a fulcrum (Fig. 6).

Associated with inversion of the stomach, eventration of the left dome of the diaphragm was seen. No attempt will be made to discuss this subject since the articles of Bayne-Jones,[2] Korns,[8] Reed and Borden[9] are exhaustive and comprehensive. Interestingly enough, in both of Ekstrom's cases, inversion of the stomach was associated with eventration of the left dome of the diaphragm and in one of Rhinehart and Rhinehart's cases the left dome of the diaphragm was absent. What part this anomaly plays in inversion of the stomach is not known. Most likely, it is a corollary of the dictum, "Where there is one anomaly, others are likely to be found."

SUMMARY

1. A case of complete inversion of the stomach is presented.

2. This case differs from all the other cases reported in that the stomach is not only inverted but reversed, the antrum and duodenal cap being situated in the left upper quadrant. In the other cases reported, the antrum and duodenal cap are located well to the right of the midline.

3. This anomaly was associated with other congenital abnormalities: eventration of the left dome of the diaphragm, incomplete descent of the cecum, and iliac ectopia of the right kidney.

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LESIONS OF THE DIAPHRAGM

WITH SPECIAL REFERENCE TO EVENTRATION AND A REPORT OF THREE CASES

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and
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THE statistical value of the summarized findings in the consecutive examination of a large number of chest roentgenograms at an Armed Forces Induction Station is unquestioned. This is particularly true when one considers certain variations from normal and minor congenital defects which often have no clinical symptoms and are therefore seldom followed to the autopsy table.

In most of the recently reported series of diaphragmatic lesions, about 80 per cent have been esophageal hiatus hernias.[5,6] It is true that these hernias can sometimes be diagnosed on the routine chest roentgenogram, but this is uncommon, and none of this type are included in this report. In fact, we must admit that not a single case of esophageal hiatus type hernia was diagnosed from the chest roentgenogram alone during the examination of this group of roentgenograms.

There were 38 cases of diaphragmatic lesions found during the examination of 412,149 chest roentgenograms. Of these, only 3 were considered from the roentgen standpoint alone to be true diaphragmatic hernias. The first of these was a congenital absence of the posterior portion of the left diaphragm. The stomach, splenic flexure of the colon, and several loops of small intestine were herniated into the thoracic cage. The second was a congenital hernia through the right foramen of Morgagni. The third was a traumatic hernia through the left hemidiaphragm following an inflammatory necrosis. The remaining 35 cases were congenital eventrations of the diaphragm, 30 occurring on the left and 5 on the right.

Since the beginning of routine roentgen examinations, eventration of the diaphragm, among other abnormalities of the human anatomy, has become rather commonplace. It is generally conceded to be a congenital condition since it has been found in the newborn. Clinical findings include an elevation of the diaphragm with a normal regular contour. Usually the normal respiratory excursion is absent and occasionally paradoxical movements are noted. Compression of the lung on the affected side is not seen and usually the condition is asymptomatic.

Pathological examinations of true congenital eventrations have been uncommon. Degeneration of the phrenic nerve secondary to trauma, tumor growth, and cervical tuberculosis have been reported and it may well be that eventration is not a true congenital anomaly, but is the result of birth injury to the phrenic nerve.[1] Degeneration of the diaphragmatic musculature is apparently secondary to destruction of the phrenic nerve.

The diagnostic roentgen signs of eventration are a high position, a regular arched contour, and possible changes in the excursions due to the defective musculature.[3] By far the greater percentage occur on the left side. In our series of 35 cases only 5 were found on the right side.

The differential diagnosis of eventration and diaphragmatic hernia is not always easy. If the diaphragm can be completely outlined and it can be determined that all the abdominal contents are below, one may be reasonably certain that he is dealing with a simple eventration. Because the elevated diaphragm is usually very thin, even this is not always possible, and some have resorted to pneumothorax or pneumoperitoneum for differentiation.[2] If air passes from the abdominal cavity to the thoracic cavity or vice versa it becomes obvious that the lesion in question is a hernia.

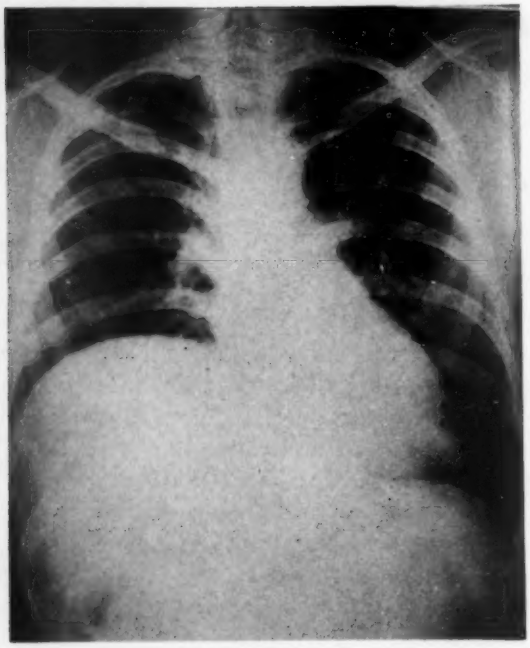

FIG. 1. Case I. Roentgenogram of the chest showing elevation of the right diaphragm to the level of the fourth rib in front. The diaphragm is regular in outline and bears the configuration of the dome of the liver directly beneath.

Marks,[7] in discussing the anatomy of the diaphragm, points out that the formation of the diaphragm and the separation of the celomic cavity into the pleural, pericardial, and peritoneal cavities is a complex process beginning in the human embryo when it is about 2.1 mm. in length. Division is not complete until the embryo is about 20 mm. in length, and the muscle fibers of the diaphragm form even later and are derived from an independent source—the fourth cervical myotome. If displacement of a portion of gut takes place before the division of the celomic cavity is complete, the misplaced loops of bowel will not be covered by a serous membrane and the hernia will not be a "true" one. If displacement occurs later, but before the muscular elements of the diaphragm have developed, the gut will

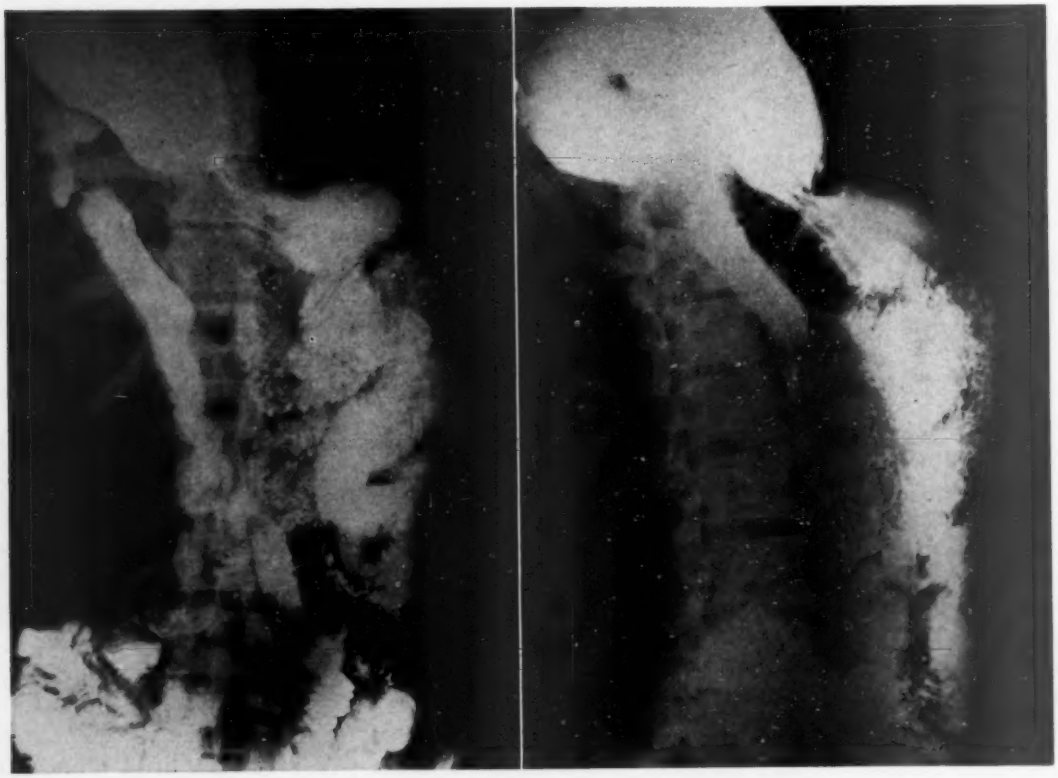

FIG. 2. Case II. Roentgenograms in the posteroanterior and oblique positions showing the body and pylorus of the stomach, and the first part of the duodenum located below the elevated right diaphragm and above the liver.

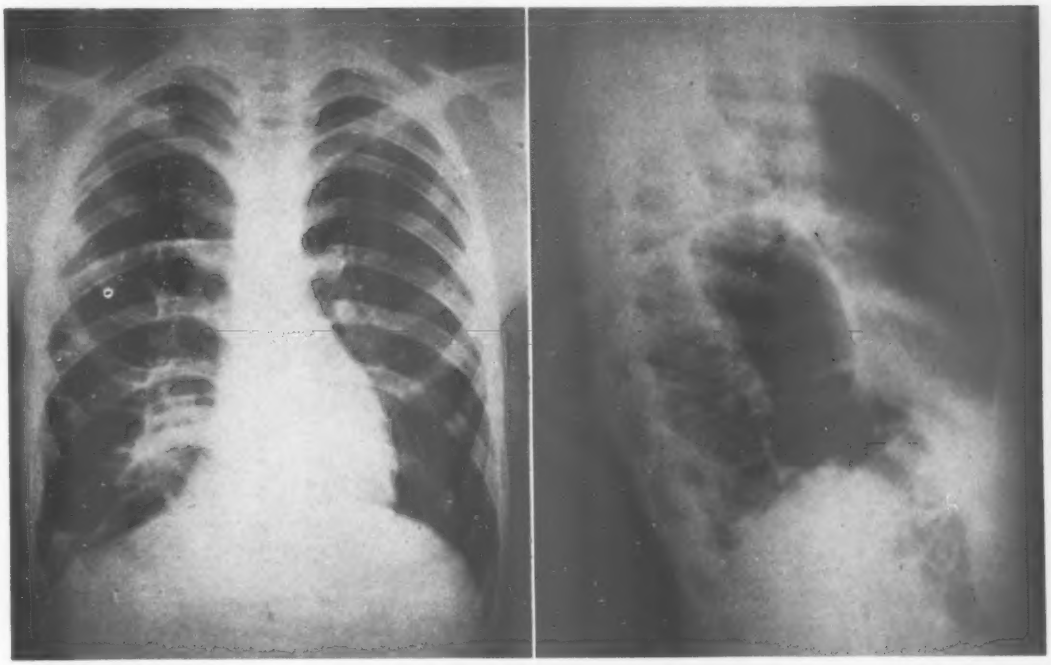

Fig. 3. Case III. Roentgenograms of the chest in the posteroanterior and right lateral positions showing elevation of the right diaphragm to the level of the second rib in front. Note the collections of gas and the bowel pattern in the right thoracic cage below the elevated diaphragm.

be covered by a serous membrane or sac and the condition will be a "true" hernia.

From the preceding discussion it becomes clear that accurate classification of diaphragmatic lesions is usually not possible. From the practical standpoint it seems to be at least necessary that we separate simple eventrations from diaphragmatic hernias. Because of the uncommon anatomical changes found in some of our cases of eventration, case histories and roentgen findings are presented.

REPORT OF CASES

Case I. A twenty-seven year old white selectee was examined for induction into the Army on April 24, 1941. He was found to be in good health and to be physically fit except for a marked elevation of the right diaphragm. Further examination and history revealed no explanation for this condition. Roentgen examination of the chest showed an elevation of the right diaphragm to the level of the fourth rib anteriorly. The liver was located directly under the diaphragm. A diagnosis of congenital eventration of the right diaphragm was made (Fig. 1).

Case II. A twenty-two-year old white selectee was examined for induction on August 26, 1943. He had no complaints and physical examination was negative except for roentgen evidence of an elevation of the right diaphragm. Further examination, including roentgenoscopy and a barium meal, revealed that the body and pylorus of the stomach, loops of small bowel, and the hepatic flexure of the colon were located directly under the diaphragm and above the liver (Fig. 2).

Case III. A nineteen year old white selectee was examined for induction on September 7, 1943, and was found acceptable with the exception of a definite elevation of the right diaphragm. The chest roentgenogram revealed what appeared to be bowel located high under the right diaphragm and above the liver. Roentgenoscopic examination substantiated this and revealed the entire stomach, as well as the hepatic flexure of the colon and portions of the small bowel, located above the liver and below the diaphragm (Fig. 3 and 4).

Although most cases of eventration are asymptomatic, the abnormality becomes of clinical significance when it results in gross displacement of abdominal organs. Cardiac

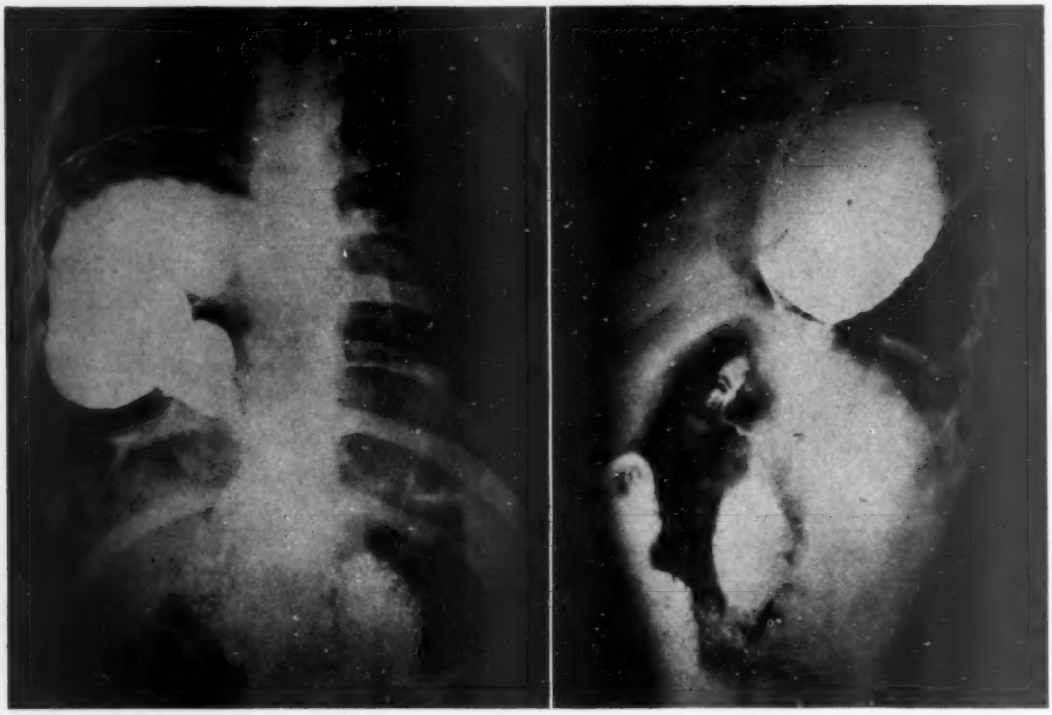

FIG. 4. Case III. Roentgenograms of the chest in the posteroanterior and right lateral positions following the administration of a barium meal. Note that the entire stomach is located directly below the elevated right diaphragm and above the liver. Later roentgenograms showed that small intestine and portions of the large intestine are also located above the liver.

displacement of more than a slight degree was not found in this series, but with an increase in age and in intra-abdominal pressure we may assume that in some cases cardiac symptoms will develop. Differentiation from diaphragmatic hernia is necessary and is sometimes difficult.

SUMMARY

1. Thirty-eight cases of diaphragmatic lesions were discovered during the routine examination of 412,149 chest roentgenograms.

2. There were only three cases of true diaphragmatic hernia.

3. The remaining thirty-five cases were congenital eventrations of the diaphragm, thirty occurring on the left and five on the right side.

4. Three cases of congenital eventration of the right diaphragm are presented with roentgen findings.

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PATENT URACHUS

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THE "Consolidated Indices" of the AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY (1903–1937) contains no reference to the roentgenographic demonstration of patent urachus or urachal deformities. Although Wyatt and Lanman[15] demonstrated a calculus in a urachus, satisfactory visualization of the complete urachal stalk has been rarely recorded in the roentgenological or urological literature. Cases of patent urachal and urachal cyst in the newborn have been previously described but these have concerned themselves essentially with the surgical treatment.[5] That patent urachus is not more frequently mentioned gives the impression that the condition is rare. In recent years, however, the condition has been recorded more frequently, especially in the urological journals,[3,4]

Garvin[6] demonstrated the displacement of the small bowel by the cystic mass caused by a urachal cyst. In the second case demonstrated by the same author, the urinary tract was visualized by means of 7 per cent sodium iodide injected through a patulous urachus. Although a bifid bladder was shown, the urachal stalk could not be seen in the roentgenogram made in the frontal position.

In the newborn where cystoscopy cannot be easily utilized and intravenous urography is carried out with difficulty, the use of the subcutaneous diodrast injection in distilled water for the visualization of the urinary tract is to be stressed.

This technique of subcutaneous urography has been described by Swick[11] and its use has been stressed by Vastine and Sampson.[12] Fluids are withheld for twelve hours prior to the examination. Contrast material (diodrast) consisting of 80 cc. of normal saline to which 20 cc. of 35 per cent diodrast has been added is then injected in the scapular area. Roentgenograms are made at ten minute intervals, the examination being completed at the end of fifty minutes.

A brief review of the literature with reference to the incidence of patent urachus shows that Cross in 1935 compiled 96 cases of anomalies of the urachus. In 1937, Herbst reviewing the medical literature of the past four centuries found 148 cases of patent urachus recorded during this long period. His painstaking work included not only the type of anomaly but also the operative procedure employed and the postoperative results that were obtained. In a total of 200,000 admissions to the Children's and Infants' Hospital in Boston, the diagnosis of patent urachus was made 3 times. Among 15,000 admissions to the Brady Urological Institute of Johns Hopkins Hospital, only 3 cases presented a patent urachus. In his critical and encompassing work of the umbilicus, Cullen tabulated 62 cases of patent urachus.

In 1930, Begg[1] tabulated 70 cases from the literature and completed exhaustive studies of the anatomy, histology and development of the urachus in the human embryo. Its derivation in common with the upper portion of the bladder was traced from the ventral cloaca. The upper end of the bladder becomes progressively narrow as the vesical gradually develops, although it preserves on a small scale its original structure and remains attached to the umbilicus. Shortly after birth, as the bladder descends taking the urachus with it, the obliterated ends of the umbilical arteries are dragged with it. The urachus with the obliterated umbilical arteries marks the upper limit of the prevesical space. It is a remnant of the embryonic allantois which is wholly vestigial in man. The intra-abdominal portion of the allantois is designated as the urachus. The allantois including the urachus runs from the top of the

bladder to its attachment to the placenta. The urinary bladder is developed as a dilatation of the distal extremity of the allantois. If the allantois remains patent, it presents itself as the patent urachus which may be an open tube from the bladder to the umbilicus or end as a blind pouch with no connection to the urinary vesical. The abdominal portion of the aliantois or the urachus situated between the peritoneum and the posterior sheath of the rectas muscles is extraperitoneal in its entire course. Begg states that the urachus extends upward from the anterior border of the bladder from a point usually 5 to 10 mm. below the apex of the vesical for a distance of 5 to 5.5 cm. The urachus, a musculotendinous structure, is situated between the transversalis fascia and the peritoneum and is movable in its lower part during the expansion and contraction of the bladder. The epithelium of the urachus may persist throughout life in numerous individuals. Hinman points out that the urachus may communicate with the bladder in one-third of all normal persons. Urine normally does not enter into the blind urachal canal, even when its open end can be probed or seen cystoscopically because of a transverse fold (Wutz's valve).

Begg[1] and Hinman[8] have described four types of anomalies of the urachus:

1. The urachus is patent throughout and is known as the complete type.
2. Patency of the urachus at its umbilical end is known as the blind external type.
3. A patent urachus at the vesical end is termed the blind internal type.
4. The ends of the urachus are closed but failure of complete obliteration of some part between the ends is present. This type gives rise to the cyst of the urachus.

Because of a deficiency of the anterior abdominal wall, the presence of patent urachus and the high attachment of the bladder in the fetus, the bladder may open at the umbilical level. Such cases have been considered as cases of simple extrophy of the bladder. Cases of congenital patent urachus may be recognized on the day of birth, but are usually noted after the umbilicus sloughs off. Often patency of the urachus may not develop until the child is several months old and may be associated with stricture of the urethra. The association of an omphalomesenteric duct and patent urachus is very rare.

Small urachal cysts are considered of little importance and may cause few or no symptoms. While most cysts vary in size from an orange to a grapefruit, others have become large enough to be mistaken for pregnancies. These cysts are characterized by the gradual enlargement of midline of the lower anterior abdominal wall between the symphysis and the umbilicus. The differential diagnostic possibilities which must be excluded are ascites, appendiceal abscess, ovarian cyst and a distended bladder. In the examination of the urachal cavity, it is connected with the bladder or the umbilicus. At times, the urachal cavity may form an accessory or storage bladder in which a cystitis may develop as in the normal bladder. At times, it may be necessary to open and drain the accessory sac and such a mass must be differentiated from a distended bladder, ovarian cyst or a subcutaneous mass.

Various roentgenographic procedures may be utilized in the examination and differential diagnosis of patent urachus. Preliminary roentgenograms in the frontal and lateral views should be first made to determine the presence or absence of abnormal soft tissue shadows, the contour of the kidney outlines and the distribution of the gas-filled bowel loops. In cases where a catheter can be introduced into the abdominal opening of the urachus, visualization of the tract by means of 3 per cent sodium iodide solution may be used. Introduction of a catheter into the bladder through the urethra followed by the injection of contrast material into the bladder should also be attempted. This should be done with great care utilizing a sterile technique in order to minimize infection. This was attempted in

our case without success. Examination of the upper urinary tract to exclude abnormalities in this region by means of subcutaneous urography has been previously stressed. In older infants where retrograde pyelography and cystography may be utilized, direct examination of the bladder should be attempted.

The following case history is of interest because of the external abdominal mass which presented a difficult differential diagnostic problem, the roentgenographic procedures which were completed and the adequate recovery of the patient following surgery.

The patient, M. G., a female infant weighing 6 pounds and 10 ounces, was delivered on November 5, 1942. The cry was spontaneous and the color of the child was good. No abdominal rigidity or distention was noted nor

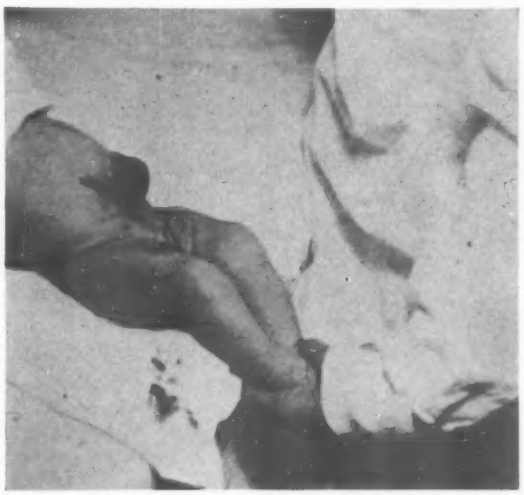

Fig. 1. The protruding cylindrical abdominal mass is shown above. The mucosa was at first thought to be small intestine in character. A wide diastasis of the rectus muscles could be palpated above the protrusion. For this reason, a persistent omphalomesenteric tract or a patent urachus associated with a congenital umbilical hernia was considered.

could any intra-abdominal masses be palpated. A well defined defect of the umbilicus with a protruding elongated mass covered with overhanging skin, the center of which contained mucosa resembling that of small intestine in character, was present. This cylindrical mass ex-

Fig. 2. A soft catheter has been introduced into the urachal opening and contrast material (approximately 10 cc.) has been injected.

tended out approximately 6.5 cm. from the anterior abdominal wall and was 3.2 cm. in diameter (Fig. 1). The examining surgical resident considered it to be a defect of the navel and a diagnosis of omphalocele was made. Roentgen examination was requested in order to determine whether or not air could be demonstrated within the mass in order to exclude a defect of the abdominal wall in which bowel was contained.

On November 12, 1942, during the examination of the patient, 1 cc. of clear yellow-tinged fluid was extruded from the center of the mass immediately after the patient had voided normally. Patency of the urachus was suggested by the type of fluid.

An attempt made to introduce a catheter into the urethra on November 18 was not successful. However, diodrast was injected by means of the catheter through the opening of

FIG. 3. Increased distention of the bladder by the dye is noted.

the abdominal mass and visualization of the bladder and the urachal stalk was demonstrated at this time (Fig. 2 and 3).

On November 27, examinations of the gastrointestinal tract were completed in order to exclude defects of the gastrointestinal tract. An upper respiratory infection at this time made it

Fig. 5. Contraction of the bladder following urination and the passage of the contrast material through the patent urachus are shown.

necessary to postpone the additional roentgen procedures. On December 7, subcutaneous urography was completed demonstrating normal pelves and calyces in the upper urinary tract.

FIG. 4. The protruding mass in the umbilical region is demonstrated. Diodrast outlines the bladder and the urachal stalk. The contrast material (35 per cent diodrast in normal saline) is seen in the posterior scapular area. The frontal views demonstrated a normal upper urinary tract.

The ureters also appeared normal (Fig. 4 and 5).

In the interim, the protruding abdominal mass reduced itself markedly in size and the patient had continued to gain so that on January 1, 1943, the patient weighed approximately 8 pounds and 6 ounces.

On January 6, operation was performed by one of us (R. W. N.). A transverse elliptical incision was made about the umbilicus as in a typical Mayo umbilical hernia procedure. The anterior rectus sheath was widely exposed about the umbilicus and then opened transversely, exposing the peritoneal cavity. Exploration showed that the bladder connected almost directly with the umbilical opening. The round ligament of the liver was seen extending upward from the umbilicus and below two yellowish cords were seen and assumed to be obliterated hypogastric vessels. The narrowed urachal stalk and portion of the bladder attached to the umbilicus were excised and removed along with the umbilicus. The open bladder was closed with a double inverting running suture of Lukens gut, and then allowed to retract to its normal position. The rectus sheaths and peritoneum were overlapped from above downwards with interrupted mattress sutures of chromic gut and the skin closed without drainage. The postoperative course was uneventful. The wound healed without any discharge and the child urinated normally. The patient was discharged from the hospital on January 18, 1943.

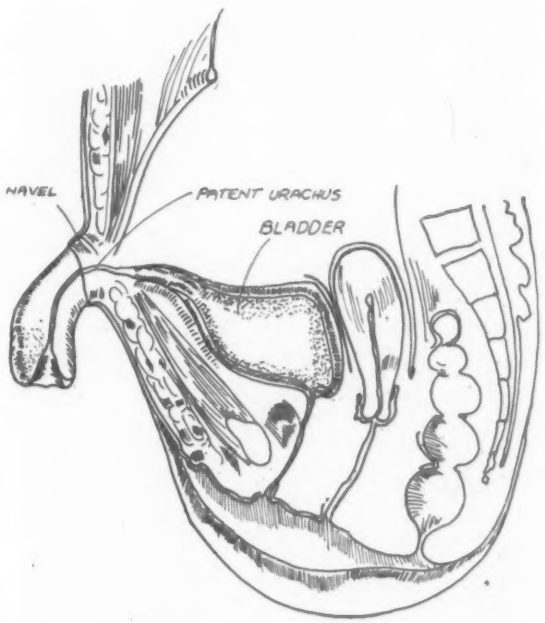

Fig. 6. Diagrammatic sagittal section through the lower abdomen demonstrating the relationship between the bladder, urachal stalk and the umbilical hernia.

The patient has been seen on numerous occasions in the out-patient department and has continued to gain weight. No urinary difficulty has been experienced at the present time.

On October 5, 1943, an attempt was made to introduce a catheter into the urinary meatus in order to demonstrate the bladder but this could not be successfully completed.

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DYSCHONDROPLASIA WITH HEMANGIOMATA (MAFFUCCI'S SYNDROME)

CASE REPORT

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Medical Corps, Army of the United States

IN 1942, Carleton, Elkington, Greenfield and Robb-Smith[1] reported 2 cases of dyschondroplasia associated with multiple hemangiomata. They were able to collect 18 additional examples after a thorough search of the literature. In almost all of the total of 20 cases thus reported the deformities were quite severe, some requiring amputation of an extremity.

According to Carleton's group, this combination of an osseous and a vascular nongenital defect was first reported by Maffucci in 1881. Although the original report was not available to them, it had been quoted at length by Torri.[2] The typical case, as described by Carleton and her co-authors, is usually a male (14 of 20 cases), who is normal at birth. During the prepubertal years small nodules, 1 or 2 cm. in diameter, appear on the small bones of the hands or feet. Later other nodules are noted, some on the long bones. The distribution is unilateral or markedly asymmetrical. Dilated veins and soft, bluish tumors become apparent in a similar distribution on the extremities and on the trunk. Fractures may follow trivial injuries and union often delayed. Development of the two sides of the body is sometimes unequal and the distortion of the normal bone structure may result in secondary deformities such as pes planus, genu valgum and scoliosis. In some cases the alterations of the hands and feet are so grotesque that the extremity is unrecognizable. The disease is stationary after full growth has been reached, but most of the patients are short and have poorly developed musculature. Intelligence is average. Occasionally malignant changes in the bone lesions may occur.

The roentgenologic findings are quite striking. The small bones of the hands and feet (but not the carpus and tarsus) are the seat of multiple small cystic areas (enchondromata), with irregular expansion of the cortex and widening of the shafts. The bones are thus quite irregular in outline. Similar areas may be seen in the long bones and occasionally in the ribs, scapulae and vertebrae. Microscopic examination indicates that the dyschondroplasia results from failure of absorption of the cartilaginous growth plate of the epiphysis. Osteochondromata may be present. Phleboliths can be seen in the hemangiomata.

The changes are undoubtedly congenital in origin, but there is no evidence that it is inherited. The association of multiple hemangiomata and dyschondroplasia is probably a coincidence. The syndrome belongs to the group of congenital mesodermic dysplasias.

The following case is an example of a milder form of this entity, and is reported because of the belief that such less pronounced cases are more common than would be supposed from the fact that only 20 instances have been reported to date.

CASE REPORT

The patient, a twenty-seven year old soldier of Italian parentage, was admitted to the Station Hospital on December 28, 1942, complaining of a "lump on the left shoulder," which was painful and tender when he wore a pack.

No member of his immediate family had ever had any similar tumors. The mass on the left shoulder, in the region of the scapula had been present for at least ten years and had not changed in size. He had sustained "baseball" fractures of the second and third digits of the left hand some ten years before his admission to the hospital, and dated the deformity of these fingers from that accident. The soft tissue tumors to be described had been present "ever since he could remember."

The patient was 67 inches tall and weighed

Fig. 2. Left wrist. In the soft tissues on the dorsomedial aspect of the distal end of the ulna there is a rounded soft tissue mass, containing several small, well defined calcified shadows approximately 1 mm. in diameter (arrows). Similar shadows are seen in a soft tissue mass at the proximal end of the fifth metacarpal (arrows). These represent phleboliths in hemangiomata.

Fig. 1. Left hand. The second and third digits are the seat of numerous small cystic areas (enchondromata), which in some places cause expansion of the cortex. The shafts of the phalanges are irregularly widened. The trabeculae are coarsened and spicules of bone arise from the cortex and extend into the soft tissues. In the distal ends of the second and third metacarpals similar cystic areas are present, as well as projections of bone from the cortex which resemble small osteochondromata. The articular surfaces are not involved. (The right hand and wrist were normal.)

Fig. 2

Fig. 3. Left foot. In the soft tissues of the fifth toe (arrow) and of the lateral border of the foot (arrows) there are a number of small phleboliths. The soft tissue margin along the lateral border of the foot is wavy, due to the presence of a series of small hemangiomata. The bones are normal. (The right foot was normal.)

Fig. 3

Fig. 4. Left ankle, lateral view. A cluster of about 30 sharply defined small (1 to 3 mm.) calcified shadows (arrow) are present in the soft tissues behind the talus. In the anteroposterior view this group of calcifications was seen to be lateral to the achilles tendon. A similar cluster of phleboliths is present below the calcaneocuboid articulation (arrow). These calcifications were at sites of the hemangiomata visible clinically. (The right ankle was normal.)

145 pounds. There was no asymmetry in development. Circumferential and linear measurements of the upper and lower extremities were equal in each case. The right side of the body was normal, both by physical and roentgenologic examination.

A number of tumors, some bony and some vascular, were present on the left side of the body. The bony tumors are described first. A low, rounded, hard, slightly tender tumor was attached to the spine of the left scapula. At the third left costochondral junction there was a hard, immovable tumor, about 3 cm. in diameter and not attached to the skin. The second and third digits of the left hand were grossly deformed and made irregular in outline by a series of bony hard, non-tender nodules. There were similar hard swellings on the dorsal surface of the left second and third metacarpals.

In addition to these bony tumors, soft tissue tumors were present over (1) the left third rib anteriorly (just lateral to the bony tumor), (2) on the dorsomedial surface of the distal end of the left ulna, (3) on the dorsum on the left hand, (4) just lateral to the left achilles tendon, (5) below the tip of the left lateral malleolus and (6) along the dorsolateral and plantar surfaces of the left foot. All of these soft tissue tumors were from 1 to 3 cm. in diameter, were attached to or within the skin, non-tender, slightly movable and in some instances could be made smaller by firm pressure. Those on the dorsum of the left hand appeared to be attached to the underlying tendons. Most of the tumors had a bluish tinge of varying degree.

The roentgenologic findings are illustrated in

Fig. 5. Left shoulder and ribs. The anterior end of the third rib is expanded and a tumor containing irregularly distributed, stippled calcification is present (arrow). This tumor measured 3 cm. in diameter and represents a typical osteochondroma. In the middle of the spine of the scapula and on the coracoid process (arrows) there are similar small osteochondromata. There is also a small cystic area in the medial portion of the neck of the humerus (arrow). (The right shoulder girdle and the remainder of the thorax were normal, as were the vertebrae, pelvis and skull. There were a few phleboliths [not shown] in the soft tissues just above the lateral condyle of the left femur.)

Figures 1, 2, 3, 4 and 5 and are described in the legends. All positive findings were on the left side of the body. Roentgen diagnoses of (1) numerous phleboliths in multiple hemangiomata, (2) pronounced dyschondroplasia in the left hand and (3) osteochondromata of the third left rib and of the left scapula were made. The patient was returned to duty on a limited service status.

Comment. This case represents a mild degree of the syndrome, which according to Carleton *et al.*, was originally described by Maffucci. At the time this patient was seen and the diagnoses made, we were not aware of the fact that this combination had been reported as a definite clinical entity. It is probable that, in a mild form, this combination is not an infrequent occurrence.

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PRESENT VALUE OF ROENTGENOLOGY IN THE DIAGNOSIS OF APPENDICITIS*

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EVEN though the roentgenologists of the world have been engaged in the study of the appendix for the last thirty-five years and although the majority have expressed their opinions either in articles or lectures, there is no doubt that recently considerable confusion has existed regarding the value of the roentgen examination in the diagnosis of appendicitis; not only between surgeons and clinicians, but also among roentgenologists themselves. Therefore, it seems advisable as well as necessary to discuss the problem in this Congress of Radiology, in order to know to what extent we, the American roentgenologists, are in agreement regarding the use of roentgenology as a diagnostic aid in these cases. Appendicitis continues to be excessively prevalent in all countries, and because of the diagnostic difficulties present in many cases plus the slight risk now involved in the surgical treatment, many patients are thus diagnosed who show no evidence of this disease at the time of surgical removal.

Of the three types of appendicitis, the acute, the chronic and the recurrent, the last two named are more frequently studied by the roentgen ray. Therefore, it is to these to which I shall refer in the course of this dissertation.

In sudden attacks of acute appendicitis the surgeon diagnoses it immediately by the patient's symptoms, without need or time for roentgen examination. It is therefore exceptional to study a case of acute appendicitis roentgenologically. In such instances, a simple examination of the abdominal cavity may be made or a roentgenoscopic study by means of an opaque enema. According to Case, the examination by opaque enema, using a dilute solution of umbrathor, must be performed with great care and under the control of roentgenoscopy in order to avoid any excess pressure which would force the substance through a perforated appendix into the peritoneal cavity. According to the same author, the surgeon can obtain valuable information following this technique, especially in cases of non-rotation of the colon (mesentery commune) when a pain in the left iliac fossa coincides with an appendicular lesion.

DIFFERENT PERIODS THROUGH WHICH ROENTGENOLOGY OF THE APPENDIX HAS PASSED

In my opinion, since 1909, when Béclère succeeded in obtaining the first roentgenogram of the appendix, until today, roentgenology of the appendix has passed through four different periods.

First Period. During the first years investigators were concerned with (1) demonstrating that the appendix could be filled with an opaque substance and could therefore be seen by roentgenoscope or roentgenogram; (2) improving the technique, and (3) searching for roentgen signs in the morphology, the evacuation, the mobility of the appendix and the localization of the pain, which would make them suspect an inflammatory condition of the appendix. The efforts of Béclère, Holzknecht, Grigorieff, Bennett, Desternes, Case, Jordan and Cohn deserve special mention. Their research demonstrated the variability of appendicular pain in different individuals, and at the same time the conclusion was reached that the appendix could be visualized in a certain proportion of cases varying from 33 to 70 per cent.

Second Period. In the second period, which was initiated by the European school,

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they began to look for demonstrative reflex signs of an inflamed appendix in the organs of the digestive system, apart from the appendix itself. Here we find the work of Aimé and Haym, Strom, Cambies, Beaumel, Jacquet and Gally, Laroche, Garcin, Pescatori, and others. This period could be called clinical-physiological, inasmuch as, following the clinical teachings and bearing in mind that the digestive organs form a harmonious composite and that a pathological condition of one part can cause disturbances in another some distance away, the function of the entire digestive tract is studied and thus the reflex gastrointestinal signs are of value in the diagnosis of appendicitis.

Third Period. Subsequently, due principally to the influence of the North American school, the importance of the distant reflex signs diminished. The direct symptoms roentgenologists observed in the appendix and the ileocecal region again became the sole supporters of the roentgen diagnosis.

Present Period. Finally, we are in a period of confusion in which some authors, such as Thomas Scholz, believe that the only sign of value in the roentgen examination is the localization of pain over the appendix. Others, following Carman's school at the Mayo Clinic, dispense with the roentgen examination in the study of chronic and recurrent appendicitis. And, although a divergence of opinion has arisen among roentgenologists, the skepticism is even greater among clinicians and especially among surgeons.

The different periods through which the use of the roentgen ray in the study of appendicitis has passed and the diversity of opinion among roentgenologists, clinicians and surgeons have two explanations, in my opinion.

First, the clinical evolution of recent years, and second, even after removal of the appendix and its histopathological study it is difficult to know exactly whether this was the exciting cause of the patient's trouble.

The Clinical Evolution in Recent Years. The glorious clinical era was headed by the French school, reaching its peak at the beginning of the present century. For diagnostic purposes it was based exclusively on the association of a series of clinical symptoms, which singly were not of great value, but which considered as a whole afforded a very reliable diagnosis. Pathognomonic signs of determined entity were not dealt with, but symptoms common to different diseases, which group themselves in one form or another, constituted the different pathological entities.

The advent of the roentgen ray and laboratory tests promoted a fundamental change in clinical concepts. New examining procedures could not be overlooked because they furnished the doctor diagnostic findings which we may call pathognomonic, such as the presence of Koch bacilli in the sputum of tuberculous patients or a niche in roentgenograms of an ulcer. For this reason, the conclusion was reached that clinical diagnosis was of no real value unless, by some means, pathognomonic signs were found that would prove the existence of definite disease.

And as roentgenology has followed an evolution parallel to that of the clinical, roentgenologists now try to confirm their diagnosis with the pathognomonic sign, forgetting that an aggregate of symptoms is still of great value in diagnostic orientation.

DIFFICULTIES ENCOUNTERED IN ASCERTAINING WHEN AN APPENDECTOMY IS JUSTIFIED

The diversity of opinion with respect to the usefulness of the roentgen examination and the importance of roentgen signs corresponds with the uncertainty of the surgeons and clinicians in determining in which cases of chronic or recurrent inflammation it is necessary to remove the appendix.

This uncertainty is due to the fact that today it is impossible to know, even with microscopic study of the appendix, if the discomforts of the patient were produced

by a chronic or a recurrent appendicitis, because nearly all appendices present microscopic lesions due to inflammation. Therefore, from what we know at present, there is no roentgen sign that can be considered pathognomonic in appendicitis.

Physiology of the Appendix. Although the appendix has no well defined physiological function, being a part of the lymphatic system it plays a part in the physiology of defense, as a producer of white blood cells. The lymphocytes and non-granulated mononuclears formed by the appendix have important functions: they capture the solid particles they find and devour the bacteria in order to protect the body against infectious disease; they excrete an antitoxin and produce oxidases and other elements which aid the digestion of fats and albumins. The appendicial fluid has diastatic properties (Rober and Josué); the appendix, like all lymphoid tissue, provides nuclear material for the various organs of the human body (Jolly and Saragea); and according to Portier's experience, the appendix of the rabbit regenerates itself, because there is hypertrophy of the lymphoid tissue of the cecum in the proximity of the resected appendix.

If the removal of the appendix does not affect the human organism, I find the explanation in the fact that the other abdominal lymphoid tissues perform its functions after the appendectomy.

As the appendix is a sac or tube that has no other opening except that which connects it with the cecum and as in this aperture there is a valve, or fold, of the mucosa (Gerlach, 1847), retention or impairment easily occurs in the entrance and exit of substances from the cecum. This anatomical characteristic that so easily provokes disturbances in the evacuation of the appendix, the fact that the appendix is the site of election in the intestine for the elimination of various microbes (Roger, Ribadeau-Dumas and Harvier, Nordwinkin), and the richness of the cecal microbic flora, are undoubtedly the reasons why this lymphoid apparatus, similar to others of the abdominal cavity, is affected so much more frequently.

Healthy and Diseased Appendices. According to Vaughan, only from 10 to 25 per cent of adults show no appendicial lesions. Méndez Lemaitre, of Bogotá, states that of 18 patients on whom a prophylactic appendectomy was performed during surgery for another abdominal lesion and in whom there had been no previous clinical history of appendicitis, only in 2 was the appendix histologically normal.

Scholz found in 46 autopsies only 2 appendices that could be considered normal, although there had been no confirmed signs of appendicitis during the life of any of the 46 patients.

My good friend and colleague, Dr. Howard Hartman of the Mayo Clinic, tells me he does not remember having seen even one report from the pathologists of that institution in which there was not mentioned some abnormality of the appendix.

Inasmuch as the majority of appendices present histological proof of inflammation —both those considered clinically healthy as well as those that have been removed because they were thought to be diseased— the logical conclusion is that the only way of knowing whether or not the appendix was the cause of the patient's discomfort lies in the disappearance of the clinical symptoms after the appendectomy.

The same thing occurs with the appendix as with the palatal tonsils, which are also lymphoid defense tissue. For one reason or another, acute or subacute inflammatory conditions occur, which pass, leaving at times macroscopic traces of the inflammation and at others microscopic.

One of my case histories illustrates this point: The patient, N. N., was examined after an attack of subacute appendicitis. As I found the appendix erect, very painful, about the size of the little finger and very long, I was not in doubt as to its pathological condition. The patient was not operated on and a few days later the clinical symp-

toms disappeared. Two months later I made new roentgenograms. In this second examination I found the appendix of normal size, perfectly movable and without pain on palpation. I have cited this case because it is unusual not to operate after a clinical and roentgen diagnosis of appendicitis, and therefore one rarely has the opportunity of performing two roentgen examinations at different periods.

Thus it is clear that no definite criterion can exist as to when surgical intervention is justified in cases of chronic or recurrent appendicitis, since histopathology justifies almost all appendectomies.

IMPORTANCE OF ROENTGEN STUDIES BEFORE APPENDECTOMY

As the surgical removal of the appendix causes no damage to the patient, since its function of defense is carried on by other lymphoid tissue in the abdomen and since the postoperative cases of adhesions or other sequelae are rare, we should reach the conclusion that in all cases diagnosed as appendicitis an appendectomy is justified, and also accept the prophylactic appendectomies performed to prevent acute attacks.

But, unfortunately, many post-appendectomy patients continue to have discomforts similar to those suffered previously.

Connell and Gibson say that in 40 per cent of 638 patients there was no improvement after operation. Coffey found no improvement in 70 per cent. Of 509 appendectomies in the Presbyterian Hospital in New York in 1922 there was no improvement in 24 per cent. These patients were then studied clinically and roentgenologically, the conclusion being reached that in only 8 there was no explanation of the symptoms, since in 17 there was some digestive lesion, 21 were suffering from neurosis to which the symptoms were attributed. Since 1923 patients have been examined by a psychiatrist before making a diagnosis of appendicitis, and from 1923 to 1925 the percentage with no improvement dropped to 14 per cent and the number of operations was reduced from 73 to 48 per year.

If more attention were paid to the careful study of the patients to be operated upon, appendectomies would decrease in number instead of increasing as is happening today, especially in private clinics. In the city of Bogotá in 1933 appendectomies represented 8.6 per cent of the total surgery performed in charity hospitals and 22 per cent of that performed on private patients. From 1936 to 1942, of 7,250 surgical interventions on private patients, 1,925 were appendectomies, that is to say 26 per cent of the total surgery.

In my statistics from the Marly Clinic I find that of 21,000 patients examined roentgenologically there were 1,738 diagnoses of appendicitis. And 793 patients, or 45.63 per cent, demonstrated some other digestive lesion along with the appendicitis.

Complementing the aforementioned and bearing in mind the scientific value of the opinions of my respected friend, Dr. Lay Martin, I deem it of great interest to quote a few paragraphs of the interesting letter he wrote to me concerning our present theme:

> Frequently appendices which are removed show evidence of inflammation. Many times it is very difficult to tell whether it is a long standing, subacute condition, or whether it is the result of a subsiding inflammatory disease. Certainly there is evidence that the appendix is diseased. However, that is not proof that the diseased organ was responsible for the symptoms of the patient. The only real proof of this is clear cut evidence that the symptoms of the patient have disappeared following the appendectomy.
>
> I have no reason to feel that the removal of a normal appendix is detrimental to the patient, but when I say this, I wish to clarify my position as follows:
>
> Many times doctors incriminate the appendix as responsible for the patient's symptoms when there is very little or no evidence that the organ is diseased. The removal of such an organ in an attempt to cure the patient is usually a most unsatisfactory procedure. Not only will the patient not be cured, he may be made worse by

the development of subsequent adhesions. If his symptoms were neurogenic to begin with, they may disappear for a time following the removal of a normal organ but they are very apt to return when the patient again finds himself in positions which are incompatible with his psyche. In other words, I believe that it is perfectly proper to operate upon a patient for the removal of a normal appendix knowing that it is a normal appendix that one is going after and that the removal of the normal appendix is a prophylactic measure designed to prevent a potential rupture some time later in life when the patient may be far removed from medical centers. The thing that I decry is the removal of an appendix on the supposition that the appendix is the basis for the patient's symptoms without careful study being given both clinically and roentgenologically under the direction of an experienced clinician and radiologist.

Roentgenology, as an aid to the clinical examination, is in my opinion the most useful and beneficial procedure to be found for the study of suspected cases of chronic or recurrent appendicitis, provided a complete examination of the digestive tract is made following a suitable technique.

In this respect Case has said: "The X ray investigation of any part of the alimentary tract must be incomplete unless it includes a careful study of the entire digestive system. The technic of the examination of the appendix is only a part of the routine which should be pursued in every gastrointestinal case, and unless circumstances prevent, no opinion is expressed on any single part of the digestive tract until the entire examination has been completed."

Technique. The technique I employ is as follows: The evening before the examination I instruct the patient to take one or two doses of tetraiodophenolphthalein, according to his weight, and the following day he comes without breakfast for the first roentgenograms of the hepatocholecystic region. If in these plates I observe any abnormal shadow in the renal-urethral area, I then take a simple roentgenogram of the urinary tract.

Immediately thereafter, I observe the patient roentgenoscopically in order to study the thorax, esophagus, stomach and duodenum. If I find any thoracic abnormality, I take roentgenograms of the thorax when the routine gastroduodenal views are taken.

Six hours later I carry out another roentgenoscopic and roentgenographic examination, being careful to study the patient minutely in anteroposterior, semi-lateral and lateral positions. At this time I take another film of the cholecystic region.

Twenty-four hours after the first dose of barium and twelve hours before the ingestion of a second dose, I perform new roentgenoscopic and roentgenographic examinations of the cecal region. I sometimes re-study the patient the following day or complete the examination with the help of an opaque enema.

Importance of the Study of the Gallbladder. The ingestion of the tetraiodophenolphthalein the evening before the examination fulfills three purposes: first, to study the gallbladder, since I have found lesions in this organ in connection with appendicitis in 17.60 per cent of cases studied; second, to be able to have a well defined image of the liver on the roentgenograms, since its shadow can be clearly seen when the gallbladder has been filled with tetraiodophenolphthalein, and third, to take advantage of the laxative properties of the phenolphthalein forming part of the iodine compound, which helps to increase the visibility of the appendix in the subsequent examinations.

The different procedures, advocated by Czepa, Cambies, Armari, Gallard, Monés, Orliansky, and others, all have the same objective of emptying the intestine, especially with laxatives, since experiments show that in this way the visibility of the appendix is increased. Chizzola demonstrates an increase of 40.8 to 58.6 per cent in the visibility of the appendix if the intestine is previously emptied, especially when magnesium sulfate is used.

With respect to the laxative and purgative effects of tetraiodophenolphthalein, I have obtained the following figures on 2,000 patients who have taken it:

	Cases	Per Cent
One evacuation before taking barium..	793	39.65
Two evacuations before taking barium.	253	12.65
Three evacuations before taking barium	179	8.95
Four to five evacuations before taking barium	160	8.00
Six to nine evacuations before taking barium	59	2.95
Ten or more evacuations before taking barium	22	1.10
No evacuations before taking barium..	534	26.70

Nor can it be said that the ingestion of tetraiodophenolphthalein causes much discomfort to the patient, because only 29.30 per cent were nauseated and 5.83 per cent had vomiting. The rest, or 64.87 per cent, experienced nothing unusual after the ingestion.

Importance of the Gastroduodenal Examination. I consider the study of the stomach and duodenum necessary because many patients attacked by appendicitis also present gastroduodenal lesions. In my statistics I find that in 1,738 patients with appendicitis, 157 also had gastroduodenal lesions.

Importance of the Thoracic Examination. Although clinically unsuspected thoracic lesions are rare, it is justifiable to prolong the roentgenoscopic examination a few more seconds in order to eliminate them, since in 1,738 patients with appendicitis I found 60, or 3.44 per cent, who also had cardiopulmonary lesions.

Importance of Repeated Examinations of the Cecum. I have insisted on the necessity of repeated roentgenoscopic and roentgenographic examinations in different positions six, twelve and twenty-four hours after the ingestion of barium, because at the same time that the colon is being carefully studied the appendix is visualized with much greater frequency. The lateral examinations are indispensable for they are the only means of obtaining a view of retrocecal appendices, so frequent in ptosis of the cecum.

Value of the Roentgen Examination.

The roentgen examination is useful for three reasons:

1. *For the Exclusion or Confirmation of Lesions in other Organs.* By performing a roentgen examination in the manner described, we can dismiss or confirm the presence of lesions in other organs, which may be the cause of the symptoms or which may coexist with an appendicitis.

Even if we were to accept, like Carman, that "when X rays exclude the stomach and duodenal lesions they have done a great service and therefore, no more can be asked," that service would be very great, since in 45.63 per cent of the patients I have found lesions in other organs coinciding with appendicitis.

The following are the statistics on 21,000 patients whom I have examined roentgenologically in my department in the Marly Clinic:

In 1,738, or 8.27 per cent of those examined, a diagnosis of chronic or recurrent appendicitis was made. Of the 1,738 cases, 880 were women, 858 were men. Adhesions were found in 100, or 5.75 per cent.

With respect to age, in 306 cases the average was thirty years, because in a grouping made in a polygon of frequencies, the arithmetical mean and the median coincide at this figure. It is interesting to note a higher frequency at forty years and at twenty-six years, as well as the fact that the frequencies occur in pairs, a lesser always followed by a greater pair.

Two hundred and fifty-eight cases of spastic colitis were diagnosed, or 14.84 per cent, many of them being an appendicular reflex. Besides spastic colitis, 793 cases were found in which the appendicitis coexisted with another disease, as follows:*

	Cases	Per Cent
Appendicitis associated with:		
amebic colitis	183	10.52
gastroduodenal ulcers	67	3.85
duodenitis and periduodenitis	90	5.16
cholecystitis with visible calculi	62	3.56
cholecystitis without visible calculi	103	5.93
cholecystic atony	29	1.66

* The total number of appendicitis cases associated with other lesions is greater than 793, because several of the patients had a history of two or more roentgen diagnoses.

poor cholecystic function	112	6.45
gastrointestinal ptosis	171	9.83
cecal-colonic ptosis	150	8.63
cardio-aortic dilatations	44	2.52
pulmonary lesions	16	6.92
utero-adnexal lesions	38	2.18
others	249	14.32

The diagnostic value of elimination is so important that those clinicians or roentgenologists who do not perform roentgen examinations to confirm or invalidate a diagnosis of appendicitis, are supporting their other clinical diagnoses by examinations of elimination with roentgenology. My esteemed colleague, Dr. Howard Hartman of the Mayo Clinic, has been kind enough to give me his opinion as to why that institution has laid aside roentgen examinations made especially to study the appendix. He states:

> We never x-ray the patient to make a diagnosis of acute or chronic appendicitis. Few people of adult years avoid some degree of inflammation in the appendix and practically every appendix that is removed routinely at operation is reported by the pathologist to be chronic catarrhal appendicitis. At times this type of appendix is capable of developing acute symptoms, and we rely upon the symptomatology of acute attack whether actually visualized by us or obtained through history to warrant the removal of the appendix. With such an attack in the history, and soreness and tenderness on pressure over the appendix, if it is sufficiently severe, we think it justifies the removal of the appendix.

There is apparently a discrepancy between Dr. Hartman's opinion and that which I was defending regarding the utility of the complete roentgen study of the digestive system in cases of appendicitis. If we bear in mind that at the Mayo Clinic routine examinations are made of the thorax, the urinary system, the spinal column, and of the digestive system when lesions of that apparatus are suspected, it is understood, although not explicitly stated by Dr. Hartman, that the clinical diagnosis was carried out without roentgen study of the cecal-appendicular region, but the other diseases that could be confused with appendicitis or coexist with it are naturally eliminated first, through the clinical examination, the laboratory and especially by roentgen examination.

2. *For the Direct Cecal-Appendicular Signs.* In the second place it must be remembered that the clinical examination gives no information regarding the location, morphology, filling, evacuation, mobility, permanent kinks or appendicular adhesions. And these details, which are so important, not only for the diagnosis of an inflammatory condition, but also for the knowledge obtained from them regarding the anatomical condition of the appendix which assists the surgeon in adopting an appropriate surgical technique in each case, can easily be procured with great precision by means of roentgen examination.

I want to quote part of a letter in which my esteemed friend, Dr. James T. Case, deals with this debatable question of the appendix. He enumerates all the direct roentgen signs looked for in the cecal-appendicular region, and thus I will be able to comment on them and discuss the value of each.

Dr. Case states:

> If the appendix fills and empties coincidentally with the filling and emptying of the cecum, and if it is freely movable and does not very accurately coincide with a point of tenderness on pressure, I consider the appendix normal, at least at the time of the examination. Of course, one may have an appendix which on a previous occasion was acutely involved in suppuration but from which the patient may have recovered, and at the time of our examination the appendix may behave normally to the X-ray examination. In such a case I merely report that at the time of my examination the appendix is normal if the conditions above enumerated are found to be present.
>
> On the other hand, if in spite of very careful examination we have failed to visualize the appendix (and it has not been removed) then we have excellent reason for assuming that it is pathological and its lumen obstructed by kinking or by obliteration. I have been able to state, for instance, that an appendix has been obliterated for its entire extent except for the proximal

1.0 cm. I have been able to show up a residual stump of an appendix after appendectomy. A fixed position of the appendix in all examinations in spite of various maneuvers suggests adhesions. In such a case one may rather safely state that adhesions are present. A retrocecal appendix is nearly invariably buried in congenital adhesions, and knowledge of this position will permit the surgeon to favorably modify his operative approach in contrast to his usual incision.

A kinked or sharply angulated appendix, appendical stasis for several days beyond the clearance of barium from the rest of the colon, or the presence of fecaliths in the appendix are all signs of appendical pathology. Appendical stasis is a menace just in the degree that the appendix retains its contents beyond the second or third day. I have seen appendical concretions of barium as long as thirty-three days after the barium meal.

Visibility of the Appendix. Let us discuss first whether a healthy appendix that has no kinks, adhesions or obstruction of the lumen is always visible in a roentgen examination.

I am convinced that the lack of visibility after a roentgen examination that is well done almost always implies the existence of a pathologic condition.

The advantage of making repeated examinations to increase the visibility of the appendix is well illustrated by the experience of Perotti and Codeca; they studied 100 normal children in several roentgen examinations. In the first they saw 61 appendices; in the second 23 more; in the third 13; and in the fourth they saw the last three. That is to say, they succeeded in visualizing all the appendices in four examinations.

In my statistics I find 226 patients in whom the appendix was not seen, from a total of 1,000 cases diagnosed as appendicitis. In other words, 22.6 per cent were not seen.

Walton and Weinstein did not see the appendix in 18 out of 152 cases diagnosed as appendicitis, which is 11.8 per cent.

The inability to visualize the appendix because of complete obstruction of the lumen represents, according to Kelly and Hourdon, 5 per cent of the appendices removed, which is in accordance with the percentage obtained by Mallory in 4,000 autopsies.

The following authors cite statistics regarding the visibility of the appendix:

Ström	70 to 80%
Wood	93%
Schnack	75%
Moore and Merritt	80%

All the data given indicate that the appendix should normally fill with the barium and that the lack of visibility after careful roentgen examinations almost always indicates that the lumen is obliterated as a result of an inflammatory reaction.

Kinks and Fixity of the Appendix. When in repeated roentgen examinations we find the appendix markedly kinked or fixed, we may be sure that a pathological condition exists and suspect the presence of adhesions that would cause the kinking or fix the appendix in a definite position.

On this point there is no great difference of opinion among the investigators. To Walton and Weinstein a persistent angulation is a direct sign of an inflammatory condition. To Deaver and Ravdin the fixity or abnormal angulation indicate adhesions.

But it must be borne in mind, as Orley says, that fixity is of importance only when it is associated with functional disturbances, especially with pain. And it must be remembered, according to Scholz, that in many instances adhesions or kinks are erroneously diagnosed by examinations technically badly executed or by erroneous interpretation of the roentgenograms.

Evacuation of the Appendix. The appendix fills at the same time as the cecum and should empty in the same manner, due to the peristaltic movements noted by Cohn in 1911 and later corroborated by White. The segmented image that the appendix sometimes presents is caused by these peristaltic movements, and today there is no doubt as to its normality. On the other hand, the retention of barium in the in-

terior of the appendix when the cecum has already emptied has been considered pathological.

Although Assmann and Merlo Gómez feel that delay in evacuation may exist in normal cases, Fedder, Chizzola, George and Gerber, Sahyoun and Oppenheimer think that it is always pathological. To some, pathology begins when the appendix does not empty at the same time as the cecum, while to others it occurs when there is a delay of two or three days. Lay Martin feels that a retention of more than ninety-six hours generally indicates that the appendix has lost its contractile powers as a result of previous attacks of appendicitis.

To me, Case strikes a happy medium when he says that appendicial stasis is a menace if the appendix retains its contents later than the second or third day. And it is clear that an appendix that retains food in its interior for many days indicates, as Vaughan says, poor drainage, and it is therefore a site for future inflammation. Retentive appendices are pathological or constitute a latent danger to the patient.

Calculi. The concretions found in the interior of the appendix may be transparent or opaque to the roentgen ray, but the first are doubtless more frequent. Such concretions or calculi are foreign bodies located in the interior of the appendix and therefore contribute by obliteration to the provocation of acute or subacute attacks. To Rolleston, Held, and Larimore, these concretions are proof of recent or old appendicular inflammation. Undoubtedly, the calculi are a contributing factor to inflammation, although it cannot be stated they are the cause, according to Vaughan. This latter author feels that the mucus secreted in the first inflammatory period combines with masses of bacteria and these form calculi which provoke the subsequent attacks.

Thus, the presence of calculi in the appendix is abnormal, because they are an important factor in future appendicitis attacks and because they often indicate that there has been a previous inflammation of the appendix.

Erect Appendix. To the direct signs pointed out, I wish to add one that in my opinion is of great value. I refer to the finding of an appendix which during roentgenoscopy gives the sensation of "erection," with increase in thickness and disappearance or diminution of the curves that normally appear.

3. *For Reflex Signs.* The third and last point regarding the usefulness of the roentgen examination is based on the value of the reflex signs in the diagnosis of chronic and recurrent appendicitis.

The appendix, when diseased, is the starting point for reflexes which are interpreted as signs of vagotonia (Butmann, Enriquez and Rouvière).

Pain. The first of these reflex signs, and to me the most important of all, is pain. Among roentgenologists there is no difference of opinion about its indisputable diagnostic value. All agree that with roentgen examination the exact locality of the pain can be found over the appendix and the other painful sites that are clinically so confusing may be discarded.

Carman has said: "Without pain, the other signs cease to be convincing," and White adds: "It is very seldom that a patient needs an operation if there is no pain when the appendix is moved on palpating it." But others, such as Fedder, say with all fairness: "Pain has diagnostic value associated with other signs, because as Walton and Weinstein have noted, when the cecum is full it normally hurts to palpate it, because of the increase in intra-colonic pressure." And according to Sahyoun and Oppenheimer, the cecum is tender in 10 per cent of normal individuals.

On looking for pain it is important to make sure that a change in the position of the appendix brings about a change in position of the pain, the previous site ceasing to be sensitive.

Therefore, I consider appendicular pain properly observed during roentgenoscopy

as a symptom of great diagnostic value, but we cannot accept it as definite proof if it is not associated with other roentgen signs.

Pylorospasm. Pylorospasm, found so often in chronic appendicitis, is confirmed by a six hour gastric retention and tenderness on palpation of the pyloric region. I therefore call it painful pylorospasm. Many authors have mentioned it, such as Oppenheimer, Walton and Weinstein, Merlo Gómez, Feldman, and others.

The epigastric pains, the nausea and vomiting observed so frequently in chronic appendicitis are explained by these spasms.

Also, it is wise to remember that at times juxtapyloric and duodenal spastic deformities appear, which lead to errors in diagnosis. It is also possible to commit this error clinically, since Poenaru and Caplesco found 14 per cent of 2,000 appendectomies with reflex symptoms that simulated gastric ulcers.

Aerogastria. Besides the pylorospasm resulting from a six hour retention and as a direct consequence of it, I have been able to prove on many occasions an increase in the size of the air chamber, accompanied by a slight ascension of the left side of the diaphragm.

The oppression and the palpitations that trouble some cases, especially after meals, almost always coincide with aerogastria.

Increase in Size and Density of the Liver. The close relation that exists between chronic appendicitis and hepatic function is clinically recognized by the symptoms of slight hepatic insufficiency and by the slight increase in the percussion area of the liver. These reasons explain why I consider as roentgen reflex signs of chronic appendicitis an increase in the density and a slight increase in the size of the hepatic shadow.

Ileal Retention. Constipation. After pain, ileal retention is the most noted roentgen sign of appendicitis. Among numerous authors who have noted it we may cite Jacquet and Gally, Laroche, Guy, Brodin and Ronneaux, Colaneri, Garcin, Smyrniotis, Chizzola, Estor, Orliansky, and others.

Ileal retention is often accompanied by cecal stasis, and it is frequently the case that the cecum remains injected after the colon has emptied, as pointed out by Fedder, and Walton and Weinstein.

Ileal retention and stasis are the roentgenological proof of constipation, which clinically accompanies nearly all cases of appendicitis.

Ileal retention has no organic cause. It is explained by atony of the last loops of the small intestine, or by spasm of the cecal sphincter (Köhler), or by spasms of the valve of Bauhin, but the first hypothesis is more generally accepted. It is felt that one is dealing with a constipation of the small intestine similar to the cecal constipation which it accompanies.

Although gastric retention and ileal retention have the same value as appendicial reflex signs, only White and a few other roentgenologists attach importance to the first. This is due to the fact that there are mistaken for ileal retention many cases in which retention has been the cause of finding the barium meal in the final part of the small intestine much later than it should be.

Ileocecal Spasms. As is natural, spastic deformities occur in the end of the small intestine and in the cecum, for these are the sites closest to the implantation of the appendix.

The ileal spasms are difficult to evaluate roentgenologically and may be taken for the anatomical lesions of a terminal ileitis. Spasms of the ileum have been demonstrated by Etienne and Piccarda, Jacquet and Gally, Pescatori, and others.

The spasmodic cecal deformities are common, but of variable aspect. They may be deformities from excess normal cecal contractions; or deformities that simulate adhesions; or spasmodic contractions of the external cecal wall with rigidity of the wall at the appendicular insertion, similar to that produced in the stomach by an ulcer of the lesser curvature; or diminution in the thickness of the inferior half of the cecum,

which gives this part an infundibular appearance; or, finally, a condition of atony, which has been mentioned by Walton and Weinstein.

But we must bear in mind that in spastic colitis there may be deformities of the cecum and of the ascending colon similar to those found in appendicitis. Thus, it is also common to observe signs of spastic colitis as a reflex of an appendicitis (Jacquet and Gally, and Huet). In some cases I have been able to verify the disappearance of the roentgen signs of spastic colitis after removal of the appendix.

The presence of signs of spastic colitis is frequent, for in my statistics I find 258 cases which coexisted with an appendicitis.

According to Balli, these images can be explained if it is remembered that the segments of the colon react by spasmodic contractions in certain areas in response to visceral-motor, visceral-sensory and visceral-secretory stimuli, especially in those sites which correspond to the different cecal-colonic sphincters. Ienino, Perotti, Guidotti, Busi and Hirsh have also found permanent contractions in appendicitis in several of these sphincters.

The reflex signs I have just pointed out, including pain, agree with the various clinical symptoms of chronic appendicitis, and in my opinion are the only ones that plainly show the existence of an inflammatory condition of the appendix. The finding of an appendix macroscopically or microscopically diseased in the course of a laparotomy, without the patient suffering discomfort from it at the time of removal, only demonstrates that that patient—as the majority of adults—had suffered a previous attack.

The proof that several of these reflex signs, even discounting pain, can lead us to make a diagnosis of chronic appendicitis is found in one of the observations made in my doctorate thesis. There had been no pain on palpation, the diagnosis was made by the other reflex signs, the macroscopic and microscopic studies of the appendix revealed clear signs of inflammation, and the digestive disturbances of the patient disappeared after the appendectomy.

CONCLUSIONS

Summing up the foregoing considerations, I believe that in the present state of our knowledge we should accept as necessary and advantageous the complete roentgen study of the digestive tract when a chronic or recurrent appendicitis is suspected clinically. There are three reasons why this investigation should be carried out:

1. Because lesions of organs other than the appendix are confirmed or ruled out.
2. Because the direct signs found in the appendicular region supply data about the morphology, filling, evacuation, location and the presence of kinks or adhesions of the appendix, which cannot be obtained from the clinical symptoms alone.
3. Because when a chronic or recurrent appendicitis is suspected, the roentgen reflex signs, such as pain, pylorospasm, aerogastria, gastric retention, ileal retention, spastic cecal-colonic deformities, and so forth, show, in accordance with the clinical symptoms, that the process of appendicitis is active.

SUMMARY

In view of the diversity of opinion prevailing today among clinicians, surgeons, and even roentgenologists as to the value of the roentgen examination in a suspected case of chronic or recurrent appendicitis, I have reviewed the various stages through which the roentgen diagnosis of appendicitis has evolved. I have reached the conclusion that this diversity of opinion is due to the change in clinical concepts during the last few years, and also to the lack of definite evidence to demonstrate that the removed appendix was, after all, the cause of the symptoms.

Nearly every adult has suffered at some time from an inflammation of the appendix. This explains why most appendices show signs of inflammation when microscopically

studied, even those that were considered more or less normal at the time of operation.

There is only one way of proving that the symptoms complained of by the patient were due to appendicitis, and that is their complete disappearance after appendectomy.

Because of our present knowledge, there can be no definite pathognomonic symptoms of chronic or recurrent appendicitis, therefore a thorough roentgenologic investigation of the digestive tract is not only useful but necessary for accurate diagnosis, for the following reasons:

1. It rules out or confirms the presence of disease in organs other than the appendix. (In 1,738 roentgen diagnoses of appendicitis, from a total of 21,000 patients examined, I found 45.64 per cent in which appendicitis was associated with some other disease.)

2. The direct roentgen signs found over the appendix and cecum give data concerning the shape, filling, evacuation, location, and kinks or adhesions of the appendix, which cannot be obtained through a clinical examination alone.

3. When a chronic or recurrent appendicitis is suspected, the indirect roentgen signs, such as pain on palpation, pylorospasms, aerogastria, ileal stasis, spastic deformities of the cecal-colonic region, and so forth, show, in accordance with the clinical symptoms, that the process of appendicitis is active.

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ROENTGEN DIAGNOSIS OF INCIPIENT CANCER OF THE RECTUM

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I. METHOD

CANCER of the rectum is not often discovered in incipient stages because it rarely causes clinical signs and symptoms until it has grown fairly large. Hence every method available should be used without prejudice in making an early diagnosis. The results of roentgen examinations have not been satisfactory. Controlled distribution of the opaque medium, on which the demonstration of gastrointestinal lesions depends in a large measure, is difficult to obtain in the rectum. Compression and palpation can rarely be applied effectively in this region, and the filling can be controlled only by regulating the inflow of the opaque fluid. This is not easy to achieve with the conventional enema technique. With the wide tubes generally used, the filling is often so massive as to obscure small lesions, or too scanty, after evacuation, to disclose sufficient detail.[4] Reflux into the ileum may overshadow the proctosigmoidal junction, in which at least 70 per cent of all benign and malignant lesions of this part of the colon are located; and the distress usually caused by rigid tubes, especially the self-retaining types, may set up motor irritation which interferes with the examination. These difficulties can be overcome by means of a few simple technical modifications which, used routinely since 1937,[4] seem to add to the value of roentgen examinations in the diagnosis of disease of the rectum and sigmoid.

Preparation. Soap-suds enemas and drastic cathartics, which may cause motor irritation,[5] should not be used. The night before the examination the patient has a light meal. Two hours later a saline enema is given. The next morning breakfast is omitted, and a second saline enema is administered two hours previous to the examination. This two hour interval is necessary lest retained parts of the enema simulate pathologic fluid levels or dilute the opaque medium. In the presence of tenesmus, discharge, and similar signs or irritation, one may use tea, a tannic acid solution, or a chamomile infusion instead of saline enemas, and atropine medication may be indicated. Painful lesions around the anus, e.g., fissures, inflamed hemorrhoids, and eczema, require the application of an ointment or/and suppositories containing a local anesthetic about one-half hour before

Fig. 1. Pressure defect caused by a distended urinary bladder. After catheterization, the rectum and sigmoid showed normal roentgen appearances.

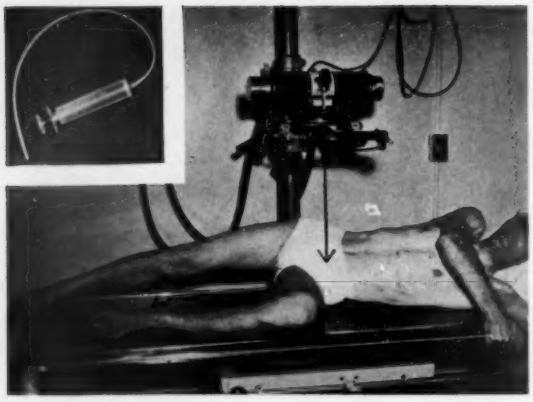

Fig. 2. Roentgenographic position for the oblique view of the rectum (see text). Insert: syringe and catheter used for roentgen examination of the rectum.

the examination is made. The bladder should be emptied, if necessary by means of a catheter, since a distended bladder may cause pressure defects in the rectum easily mistaken for tumor (Fig. 1).

Roentgenoscopy. A flexible urethral catheter is inserted into the rectum. The opaque medium is injected through the catheter with a 20 cc. syringe under roentgenoscopic observation while the catheter is gently withdrawn towards, but not beyond, the internal sphincter. According to the degree of filling required in each case, opaque medium may either be added or withdrawn with the syringe. Spot roentgenograms may be made in various positions. Once the rectum and sigmoid are studied, the examination is completed by filling the whole colon. The catheter is left in place and is connected with an enema device. With some practice it is possible to regulate the flow of the opaque medium from the enema device so accurately as to make the use of a syringe unnecessary; but by adopting the syringe method first, the examiner will make himself more familiar with the technique. In most cases, 20 to 60 cc. of opaque medium are needed for the rectum and sigmoid, and 1 to 1½ pints for the entire colon. Air may be insufflated if necessary.

Roentgenograms. In addition to spot films, roentgenograms may be made in various projections according to the preferences of the examiner. The following positions are useful: (1) The patient is supine; the tube points downward with a caudal tilt

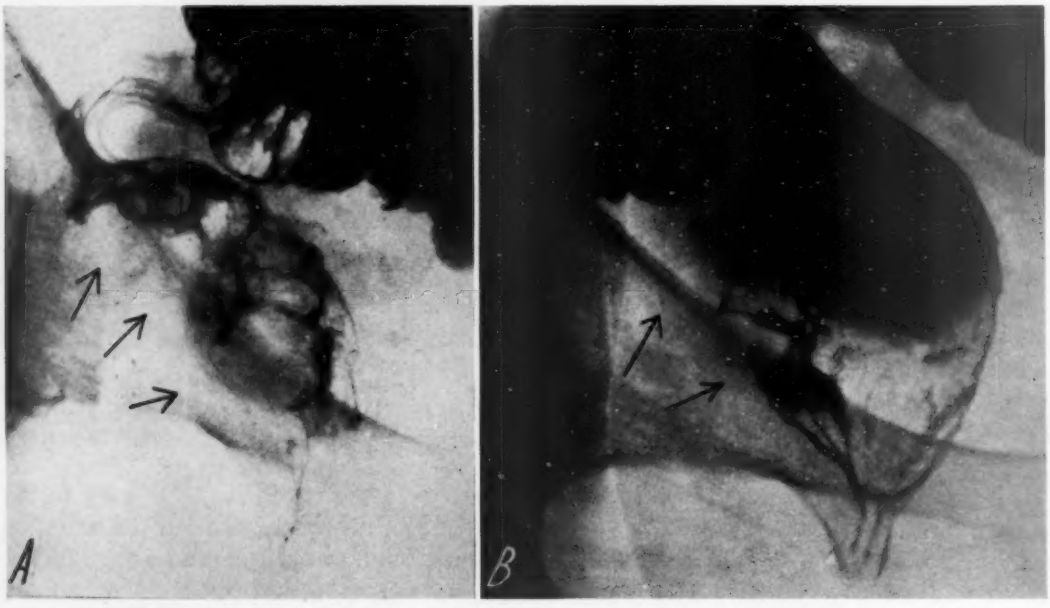

Fig. 3. Papillary type of cancer of rectum at various stages of filling. This illustrates how easily the distribution of the opaque medium in the rectum can be controlled.

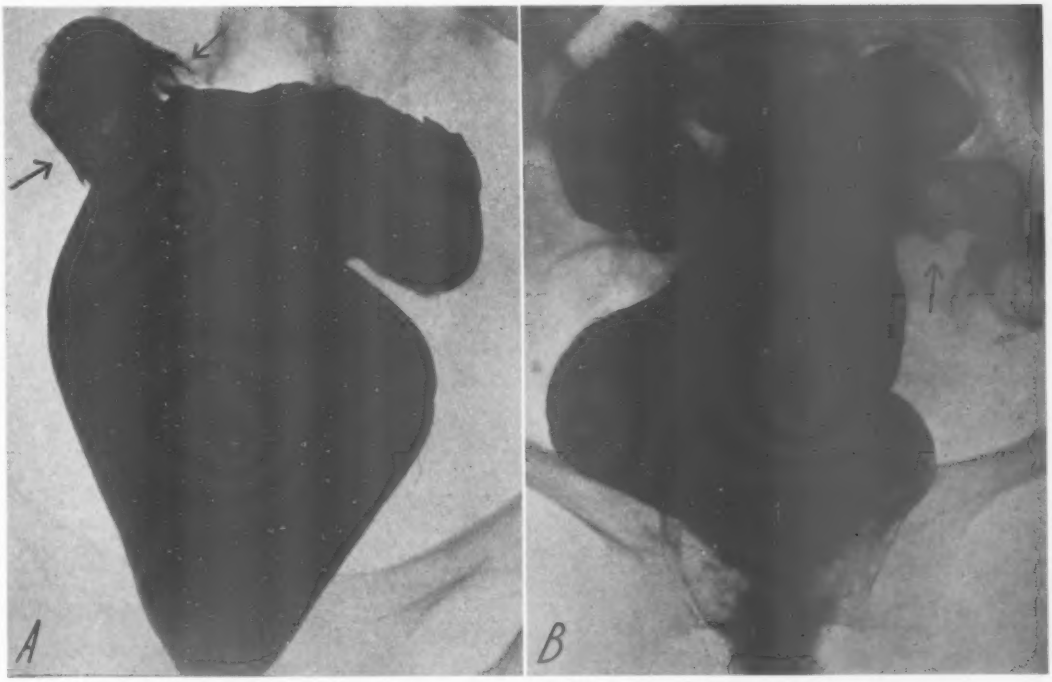

Fig. 4. During massive filling by means of the conventional enema technique (*A*), retrograde intussusception of the constricted segment takes place (arrow points to the characteristic filling defect resembling the figure 3). By means of the catheter method, the obstruction is overcome (*B*), and a small cancer crater is clearly shown (arrow).

of about 12 degrees and is centered on the midline about 1 inch cephalad of the pubic symphysis. Since the rectum is closer to the posterior than to the anterior border of the pelvic cavity, this projection usually produces good definition. (2) The patient is supine but with the right side raised by about 45 degrees; the tube is directed vertically downward to a point about midway between the umbilicus and the left inferior iliac spine (corresponding to McBurney's point, but on the left side); the left leg is bent at the hip joint, and the right leg is extended and brought backwards as far as possible, in order to avoid obscuring of the rectum by the summation of the shadows of both thighs (Fig. 2). In this position the rectum and sigmoid are nearly parallel to the plane of the film.

Comments. An enema is not made more pleasant when taken in an ill-lit room, on a hard table, and in the presence of a stranger armed with a leaden glove. Making the patient feel comfortable is perhaps the most important single factor in obtaining satisfactory diagnostic results.[1,4] Using effective cleansing measures without causing irritation of the bowel is almost equally important. The only disadvantage of the catheter method is that the catheter has to be held in place during roentgenoscopy. The main advantages are: (1) the flow and distribution of the opaque enema can be controlled so accurately that degrees of filling ranging from slight mucosal coating to massive distention of the rectum can be obtained (Fig. 3); (2) there is no distress even in the presence of painful lesions of the anus and rectum; (3) stenosis is usually overcome by the slow and regulated flow of the medium through the catheter, while it is often accentuated during massive filling, since the sudden pressure of the opaque medium upon a narrowed segment may cause retrograde intussusception and complete occlusion (Fig. 4).

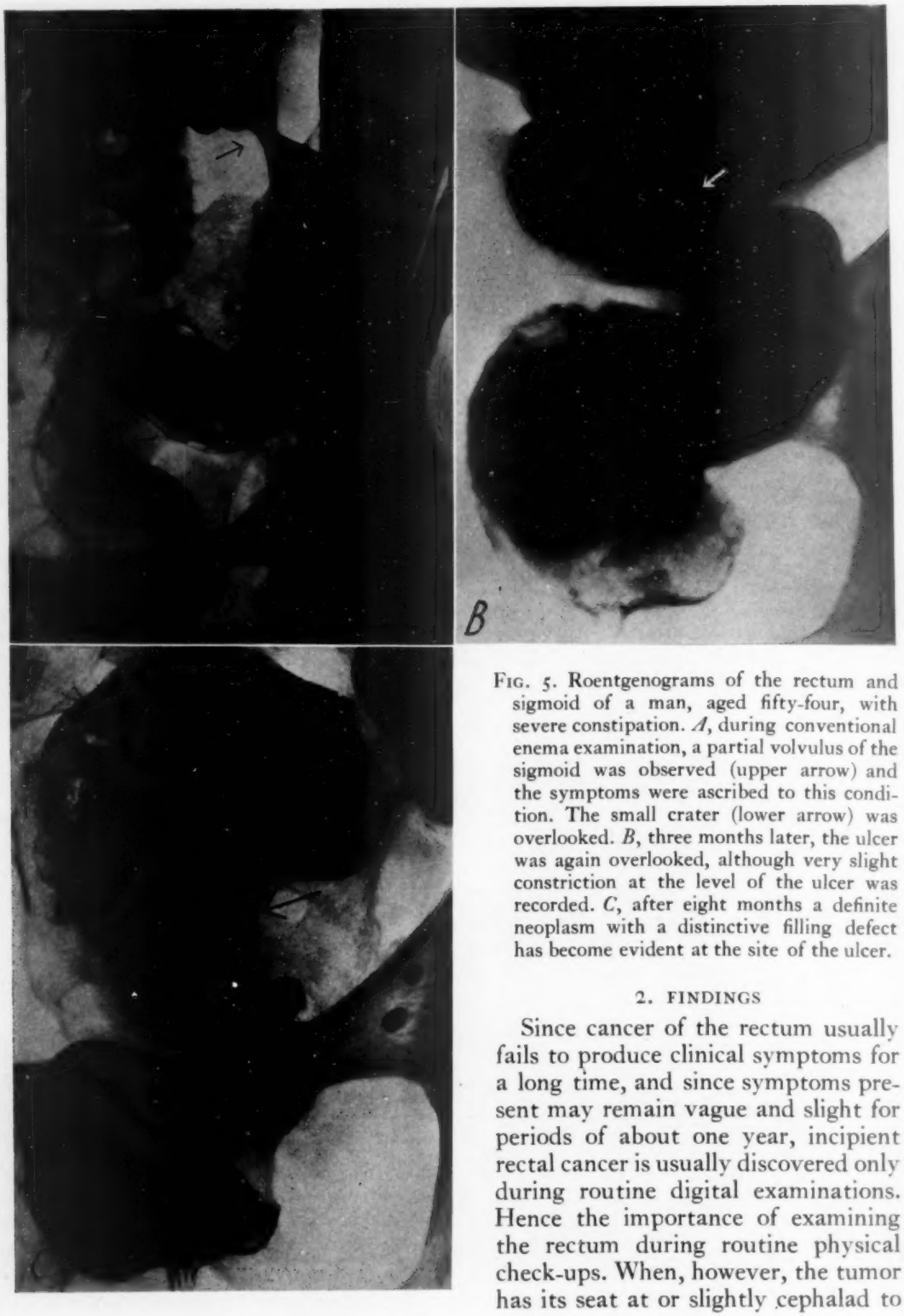

Fig. 5. Roentgenograms of the rectum and sigmoid of a man, aged fifty-four, with severe constipation. *A*, during conventional enema examination, a partial volvulus of the sigmoid was observed (upper arrow) and the symptoms were ascribed to this condition. The small crater (lower arrow) was overlooked. *B*, three months later, the ulcer was again overlooked, although very slight constriction at the level of the ulcer was recorded. *C*, after eight months a definite neoplasm with a distinctive filling defect has become evident at the site of the ulcer.

2. FINDINGS

Since cancer of the rectum usually fails to produce clinical symptoms for a long time, and since symptoms present may remain vague and slight for periods of about one year, incipient rectal cancer is usually discovered only during routine digital examinations. Hence the importance of examining the rectum during routine physical check-ups. When, however, the tumor has its seat at or slightly cephalad to

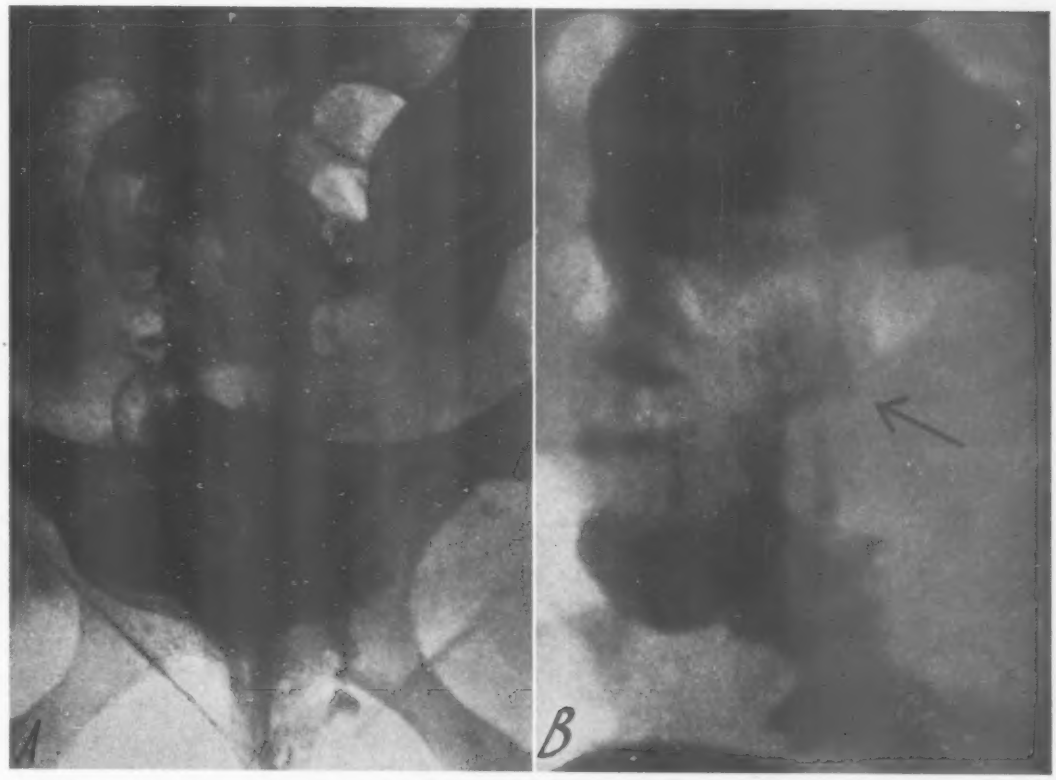

Fig. 6. *A*, anteroposterior, and *B*, oblique projections, showing a cancer crater close to the proctosigmoidal junction (confirmed histopathologically).

the proctosigmoidal junction, it may escape the palpating finger, and proctoscopy is then omitted in most cases. A certain number of cancers of the proctosigmoidal junction are not accessible to the proctoscope. For all these reasons, it is good practice to make a study of the rectum during routine roentgen examinations of the digestive tract. The additional time spent rarely exceeds five minutes. A number of cancers at early stages were discovered in the rectum in this way before being suspected clinically.

The roentgenologic appearances vary with the anatomical nature of the tumor. The large majority of rectal cancers are of the annular ulcerating type; next in incidence are cancers arising in polyps; papillary cancers are relatively rare. All age groups are involved. In this series the incidence was highest between the ages of forty and fifty-five years and almost the same in males and females.

1. Annular Type. The appearances observed at early stages do not conform to those of more advanced annular cancer. Filling defects and shelf formation are rarely present, and constriction is often so slight as to remain unnoticed, if present at all. The involved segment is easily distended during massive filling, and the absence of constriction and deformity during massive filling was responsible for diagnostic failures during the early stages of this study (Fig. 5).

There is, however, one constant sign, namely, a small ulcer with rigid walls. The ulcer produces appearances very similar to those of peptic ulcer of the duodenum. It is seen as a persistent opaque spot 1 to 2 mm. in diameter when projected endwise, and as a thorn-shaped prominence of the contour

when projected edgewise, with the thorn pointing outward. The lesion is not definitely intraluminal, and the appearances do not correspond to the meniscus sign of early gastric cancer, except at a more advanced stage (Fig. 8). The mucosal pattern may appear to be erased in the immediate neighborhood of the ulcer (Fig. 4, 5, 6 and 7).

As the tumor grows, constriction becomes evident, and there may be nodular prominences encroaching upon the lumen (Fig. 5). The findings observed at these more advanced stages are well known and need not be described here.

Among the last 52 proved cases of annular rectal cancer of this series, an ulcer was seen roentgenologically and confirmed pathologically in 50 patients; the lesion escaped roentgenologic demonstration in 2 instances; and there were associated signs of tumor, such as shelf formation, constriction, and filling defects, in 11 cases. There was often a remarkable similarity of the anatomical and roentgenologic appearances.[4]

The term "annular," used in textbooks, is somewhat misleading. These tumors are annular at advanced stages, when they involve the whole circumference of the rectal wall; but they are definitely not ring-shaped, or annular, at incipient stages, when they are confined to only a sector of the rectum. These early lesions always show a central ulcer; and although the crater is merely the earliest roentgen sign, but not necessarily the earliest anatomical evidence, of this type of carcinoma, the consistent presence of an ulcer in these cases would seem to justify the term "ulcerative" carcinoma, rather than annular carcinoma.

2. Papillary Type. There were only 3 cases in this series, and none of them was very early. The papillomatous shape of the tumor is readily recognized by the scalloped irregular filling defect which seems to arise from a circumscribed area, usually in the lateral wall of the rectum, without producing obstruction. Ulcers may be present in

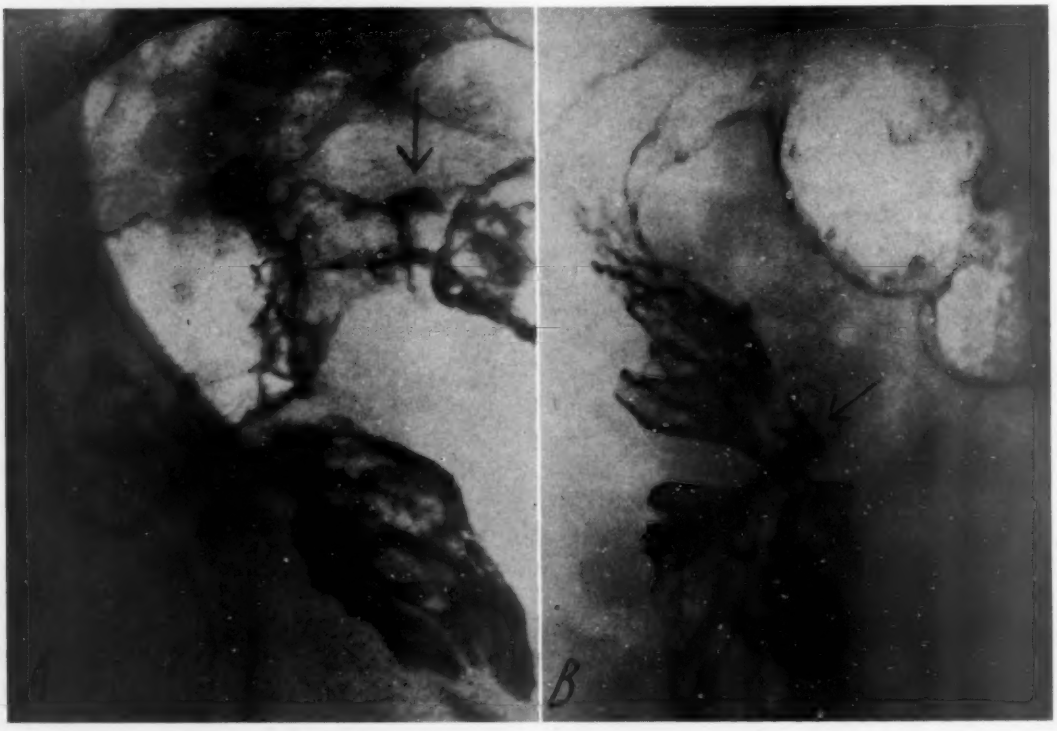

FIG. 7. *A*, cancer crater at the proctosigmoidal junction. There is slight constriction of the involved area. *B*, small crater at the site of an anastomosis made two years previously for cancer of the proctosigmoidal junction. The crater was found to correspond to a recurrence of the cancer.

these cases (Fig. 3). Owing to the absence of constriction, the involved segment remains flexible for a long time, and is readily distended during massive filling. When these tumors are located along the posterior wall of the rectum, they are easily overlooked.

4. Carcinomatous Polyps. Polyps may occur either in the form of polyposis or as single polyps scattered thinly over a part of the pelvic colon. Polyposis is easily recognized by the characteristic chain of filling defects which it produces.[4] Single polyps in the rectum are difficult to see roentgenologically. True polyps, which are neoplasms, cannot be distinguished from so-called secondary polyps, which represent localized hypertrophy of the mucosa as a result of chronic inflammation. Since a majority of true polyps prove to contain foci of carcinoma histopathologically, it is important to discover these tumors before they have become frank cancers.

Single polyps cannot always be recognized by palpation and proctoscopy since they may become buried between loose or large mucosal folds. In 4 cases, single polyps, not previously disclosed by digital examination and proctoscopy, were revealed by roentgen examination and subsequently confirmed by proctoscopy and biopsy; all of them were malignant (Fig. 9*A*). The rounded filling defects caused by polyps, easily recognized with slight filling of the rectum, are usually invisible during massive filling (Fig. 9*B*). The outlines are smooth and clean-cut, no matter whether the polyp is benign or histopathologically carcinomatous, and it is not until infiltration and ulceration set in that the malignancy of the lesion becomes evident roentgenologically.

3. DIFFERENTIAL DIAGNOSIS

When the lesion involves the ampulla of the rectum, a differential diagnosis can be made by proctoscopy and biopsy in many cases; moreover, there are few diseases which have to be considered. Internal hemorrhoids may cause filling defects resembling polyps; but the worm-like shape of

Fig. 8. Moderately advanced annular cancer of the proctosigmoidal junction, showing a central ulcer (arrow) with tumor-shelf formation on both sides.

the filling defect is usually revealed by views taken at various angles.[4] Villous proctitis, a relatively rare condition, may cause similar appearances but involves a much larger area than does cancer.[4]

Cancer of the proctosigmoidal junction is often inaccessible to proctoscopy and biopsy, and the fact that benign lesions of this region are common adds to the diagnostic difficulties. Both benign and malignant diseases of this segment tend to cause constriction and pericolitic adhesions with deformity and rigidity of the walls. The crater produced by incipient cancer may become lost in the irregularity of the contours; on the other hand, a tent-shaped adhesion may simulate a tumor crater, especially in a constricted area (Fig. 10*A*). Suppurative pericolitis may produce appearances strongly resembling those of cancer, since the pressure defects caused by small intramural or pericolitic abscesses obliterate the mucosal pattern and pro-

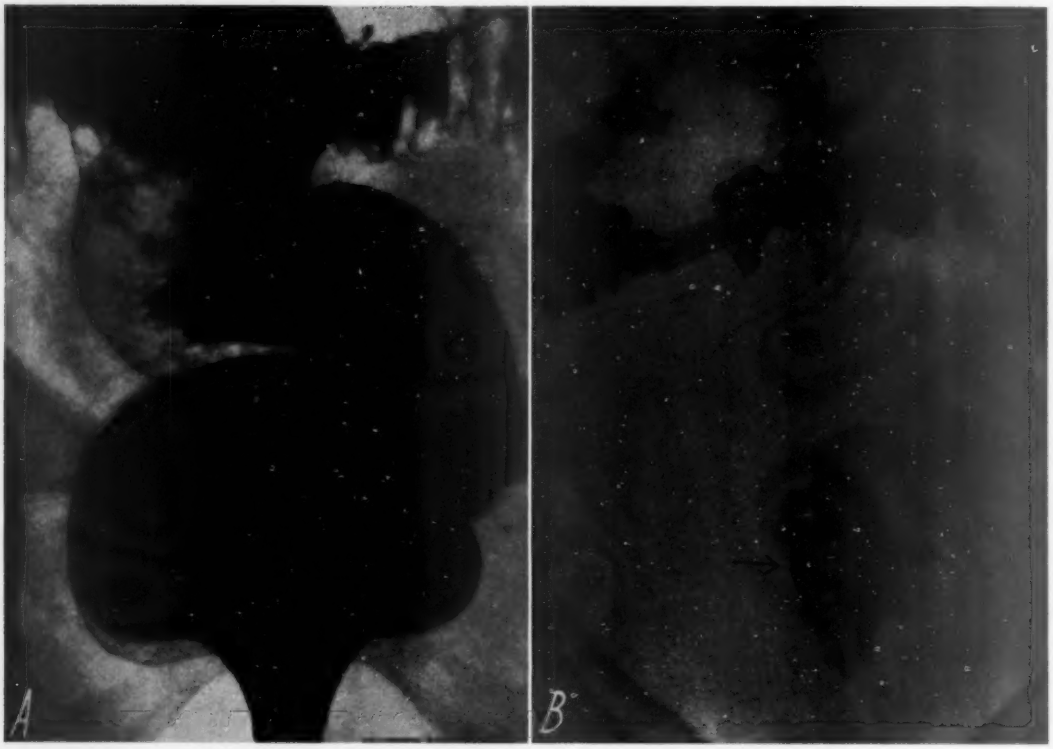

Fig. 9. Polyps of the rectum, histopathologically malignant. *A*, the polyps are not seen during massive filling with the conventional enema technique. *B*, the polyps are well shown with the modified method. These polyps had not been discovered previously during proctoscopy because they were hidden between large mucosal folds.

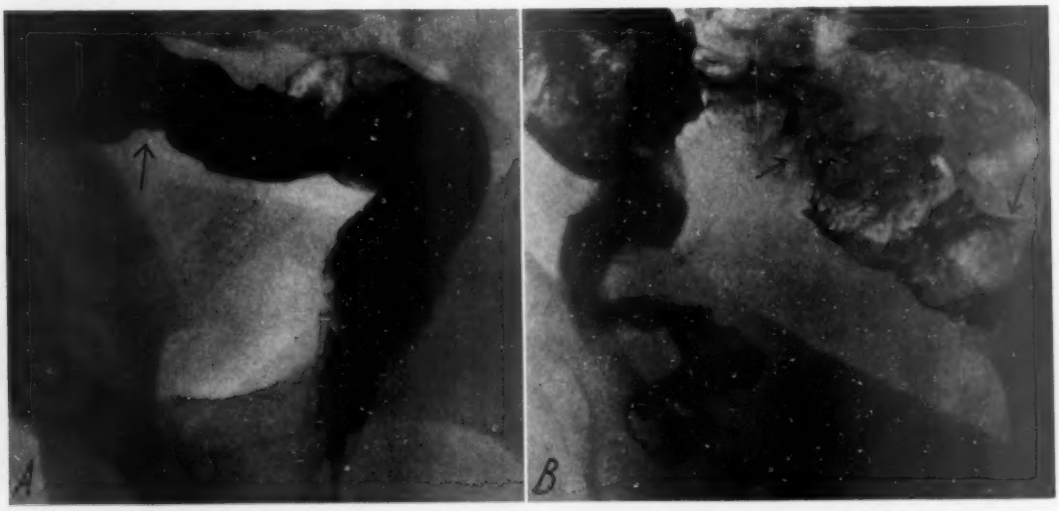

Fig. 10. Suppurative pericolitis. *A*, a small adhesion (arrow) simulates a tumor crater. *B*, multiple filling defects (arrows) caused by small pericolitic abscesses in the sigmoid.

trude into the lumen like tumor nodes (Fig. 10*B*). If there are normal areas between these formations, cancer can usually be ruled out. Since in the presence of suppurative pericolitis surgical operation involves the risk of setting up diffuse peri-

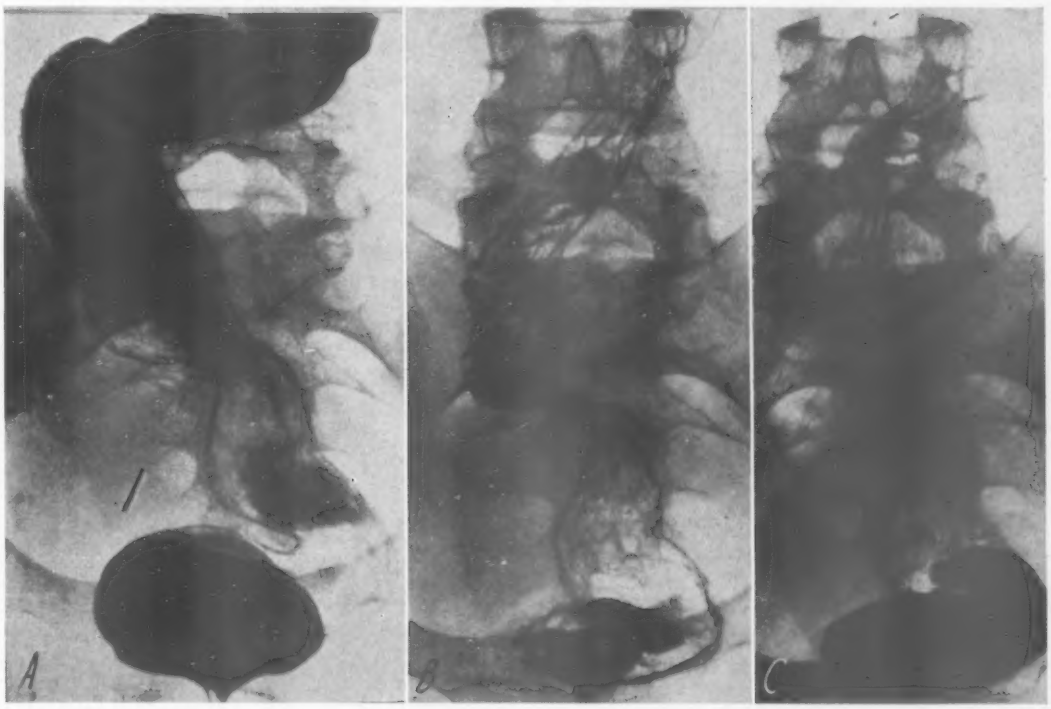

FIG. 11. Constriction of proctosigmoidal junction caused by colitis. *A*, during massive filling, and *B*, with the modified technique, a persistent narrowing of the proctosigmoidal junction is disclosed. *C*, one month later, after dietary treatment, the constriction has disappeared, and the mucosal relief of the sigmoid, coarse and irregular in *B*, has become normal.

tonitis, and since pericolitis, unless it causes obstruction, rarely requires surgical intervention, it is important to differentiate the condition from tumor.

Subacute and chronic inflammation of the proctosigmoidal junction, with or without diverticulitis, differs from cancer by the presence of persistently thickened mucosal folds.[2,4] Sometimes a circular constriction of the colon is present in these cases and subsides when the inflammation heals (Fig. 11). Stenosis of the rectum due to venereal lymphogranuloma can hardly be confused with tumor.[3]

In elderly people the distal end of the rectum often bulges concentrically around the internal sphincter, thereby producing, so to speak, an "internal prolapse."[4] The internal sphincter then stands out as a deep circular constriction which, together with filling defects caused by hemorrhoids, may produce appearances suggestive of annular tumor (Fig. 12).

4. CONCLUSIONS

The factors involved in improving diagnostic results are not solely technical. For instance, when incipient cancer of the rectum has been detected roentgenologically in a number of cases, the resulting increased interest of physicians and surgeons leads almost inevitably to a more thorough search for these tumors during routine physical examinations. Consequently the significance of roentgen examinations in the early diagnosis of cancer of the rectum is difficult to evaluate; but the fact that in a group of patients cancer of the rectum was discovered during roentgen studies of the digestive tract before being recognized by other clinical methods suggests that roentgen examination has its place in the early diagnosis of this disease. This does not by any means imply that roentgen examination is superior to digital and proctoscopic examinations. The point is that cancer of the rectum remains clinically silent for a

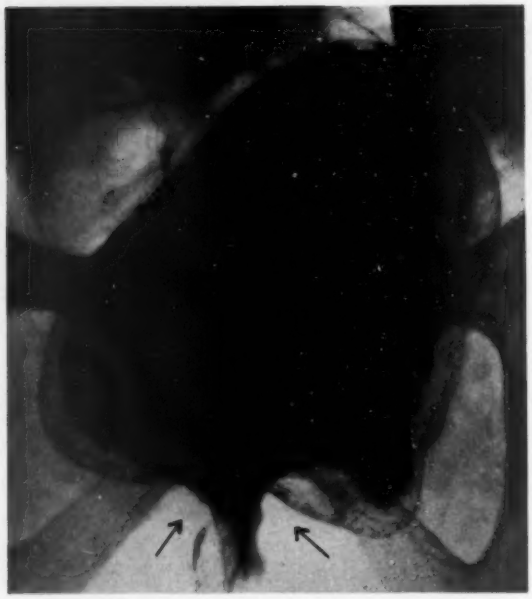

FIG. 12. "Internal prolapse" of rectum around internal sphincter in a woman aged sixty-three. The lower part of the rectum overhangs the internal sphincter (arrows). This may simulate annular constriction. See text.

long time, wherefore its incipient stages are usually discovered almost accidentally, during routine examinations. One of these routine examinations is roentgen study of the digestive tract. Consequently the roentgenologist should make an adequate study of the rectum during every examination of the digestive tract. The importance of this rule is illustrated by the fact that in a small community, with a 100-bed hospital, cancer of the rectum has been demonstrated roentgenologically at an average of 1 case in every nine days during a period of two years.*

Perhaps the roentgen findings obtained with the modified enema technique above described can be duplicated with the conventional opaque enema methods; but the modified method seems to disclose significant details with greater ease and accuracy. Whatever method is used, adequate cleansing of the bowel, prevention of motor irritation of the rectum, and an intelligent approach to the patient are important factors in obtaining satisfactory diagnostic results.

* All the cases of this series were verified histopathologically. Most of the histopathologic examinations were made by Dr. Ralph E. Miller, professor of pathology, Dartmouth Medical School; and in 2 cases, by Dr. Tracy B. Mallory, pathologist, Massachusetts General Hospital.

5. SUMMARY

1. Roentgenologic demonstration of small lesions of the rectum is facilitated by the use of an opaque enema technique so modified as to achieve accurate control of the distribution of the opaque medium in the rectum and sigmoid.

2. The most constant sign of incipient annular cancer of the rectum is a small ulcer with rigid walls. Other signs of tumor, such as constriction and shelf formation, are rarely present at early stages.

3. Malignant polyps and small annular cancers, not previously noted during digital and proctoscopic examinations, were disclosed roentgenologically in a number of instances. This suggests that roentgen examination with an adequate technique has its value in the early diagnosis of these tumors.

4. Roentgenologic appearances simulating tumor may be produced by suppurative pericolitis.

5. Since cancer of the rectum usually remains clinically silent until it has reached a relatively large size, incipient stages are discovered mainly during routine examinations. Consequently routine roentgen studies of the digestive tract should include an accurate examination of the rectum in every instance.

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CLINICAL AND ROENTGEN ASPECTS OF IRRADIATION STRICTURE OF THE RECTUM AND SIGMOID

ITS COURSE AND TREATMENT*

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BOWEL damage following irradiation for cancer of the cervix has by now been sufficiently described in the literature to be clearly established as an entity. Corscaden, Kasabach and Lenz,[1] Todd,[2] White,[3] Martin,[4,5] Chydenius,[6] Aldridge,[7] Wigby,[8] and others, have taken up variously the surgical findings, barium enema and proctoscopic appearances, related dosage techniques, pathological findings, possible methods of avoidance, and estimates of incidence of the condition. The greater number of these topics will be omitted from the present discussion, which will be limited to a functional study of types and degrees of damage of the bowel, our methods of handling the cases, and the results of treatment. We are concerned in presenting particularly the non-operative management of this condition, which in its severer forms has in the past been for the most part treated surgically.

Our material consists of 44 cases of temporary or permanent stricture of the bowel, in all but a few instances confined to the pelvic colon and rectum. Forty-two of the cases originated in the Woman's Hospital. Aldridge has reported on this material from the points of view of a general description of the condition, statistics as to occurrence and outcome, irradiation and surgical techniques, surgical-pathological findings. Those interested in this omitted background of our cases, including details of irradiation techniques, are referred to his paper. A brief discussion of incidence, though covered in the Aldridge paper, seems indicated because it bears on the nature of our material. He reported 16.9 per cent of bowel injury cases in the group studied, which is higher than the other series reported, and he described 29 cases of temporary or permanent stricture, of which 11 were operated upon. Since his paper was published we have added 15 more cases, of which only 1 has been operated upon. We feel sure that quite a few of our cases, among those designated as in Groups I and II, would not have been discovered if we not been particularly engaged in looking for them, and that otherwise our incidence would have been much lower. From subsequent experience we also believe that operation could have been avoided in several of the 12 cases operated upon, for reasons to be developed below, and this would have lowered our operative percentage. Wherefore we feel that statistics of incidence mean very little in this condition, unless barium enema studies and proctoscopies are done on every case with the slightest degree of diarrhea or other bowel complaint, which is obviously impossible of realization. A stricture relatively or entirely symptomless may be discovered, often accidentally, after five or eight or twenty-three years, as happened among our cases. A severe degree of narrowing may disappear completely within a year, while another case, with only mild bowel complaints for several years, may suddenly precipitate into acute obstruction, though this delayed outcome is rare. Further, a comparison of complications between clinics recognizing this entity and those which do not is misleading. All clinics reporting on cervical cancer note bowel complications. For example, Sackett[9] in a study of compli-

* From the Clinics of Gastroenterology and Roentgenology, Woman's Hospital, New York. Read in part before the Forty-Third Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 15–18, 1942.

cations in 688 cases of cervical cancer treated at the Woman's Hospital from 1919 to 1935 lists the following:

	Cases	Per Cent
Intestinal-vaginal fistula	60	8.7
Intestinal obstruction	25	3.6
Rectal disorders	42	6.1

He admitted that there was overlapping in these groups. Some of the rectal disorders with diagnoses founded on severe diarrhea, rectal bleeding, tenemus, cramps, and so forth, might well pass over into obstruction or fistula. But we believe that, if metastasis of the bowel is excluded, all or nearly all of the remainder of the above cases would at some time in their course have shown pictures similar to those described here. Practically all of the cases here to be discussed were treated subsequent to 1935, and do not overlap those in his report, and it is interesting to note that in our material fistulae have almost disappeared, suggesting that prompt treatment of a local focus of injury aids in preserving the blood supply and healing the ulceration, and hence in preventing development of fistulae. We feel strongly that the apparent upsurge of intestinal stricture, whether or not related to specific techniques, is no new entity, but merely one more sharply diagnosed. Cancer is a lethal disease, and its treatment, no matter how carefully done, cannot always be harmless to surrounding structures, as all of us know who are confronted from time to time with permanent skin damage, lung fibrosis, necrosis of bone and cartilage, to name but a few sequelae of irradiation of cancer in other parts of the body.

RELATION BETWEEN CURE OF PRIMARY DISEASE AND BOWEL DAMAGE

In general, the management of bowel damage tends to be a problem of the cured case,—cured, that is, of local cancerous disease. This point was made in respect to irradiation ulcer of the bladder, another casualty of cervical irradiation, by Dean in a symposium at Memorial Hospital on bladder and bowel injuries following pelvic irradiation. In his experience the bladder ulcer of this type was most frequently found in a pelvis free of cancer, the assumption being that irradiation heavy enough to injure the bladder was likely to cure the cancer. This reasoning cannot of course be applied too literally, as many cured cases do not have ulcer of the bladder, or intestine, and some visceral damage is due to faults of technique, adhesions and other causes than the mere fact of irradiation. Nevertheless there is a trend in our series suggesting that damaged bowel tends to occur in cases in which the pelvic disease clears up following irradiation and does not recur, at least for the periods covered by our study. If this is substantiated by longer observation, it makes the matter of diagnosis and treatment doubly important. If a patient is to live free from her cancer, but troubled by a bowel disorder, she does not consider the outcome successful even though she appears on the five year cure list of her particular clinic. In reading over old charts from the group cited by Sackett in his Complications Report we were impressed by the probability that at least some of these patients dying of intestinal hemorrhage or obstruction, assumed to be due to the spread of cancer, actually had hemorrhage from irradiation ulcer, or obstruction from

TABLE I

STATUS OF PELVIC CANCER IN CASES WITH BOWEL DAMAGE

1.	Cases living		29
	No clinical evidence of pelvic cancer (Cases under one year—3)	28	
	Pelvic cancer present	1	
2.	Cases dead		15
	No evidence of pelvic disease at death	11	
	Pelvic cancer present	4	
3.	Causes of death where there was no pelvic disease		
	Remote metastasis	4	
	Hemorrhage from ulcerated bowel	2	
	Postoperative peritonitis	2	
	Starvation	1	
	Cardiovascular disease	2	
4.	Total cases followed over one year		41
5.	Primary regression of cervicopelvic cancer		36

irradiation stricture, together with peritonitis from perforation of a non-malignant ulcer. Table I shows the basis of our belief that bowel damage tends to be accompanied by success in the immediate treatment of the local cancerous disease.

Thus, in 41 cases followed over one year, 1 is alive with pelvic cancer, and 4 died of it. Thirty-six cases with bowel damage, or over 90 per cent of the group, showed successful regression under treatment and with 1 exception remained free of demonstrable pelvic disease to the present. This percentage of course has nothing to do with five year survival rates, as, at the time of preparation of this table, only 9 cases had been followed more than four years. It is offered merely as an index, in an admittedly small group, of a possible relation between bowel damage and successful local treatment of the cancer.

LOCATION OF BOWEL TRAUMA

Contrary to the experience of some workers, we have had no cases of massive widespread ileal damage. This is undoubtedly a product of heavy roentgen dosage, almost invariably through large portals involving large cubic areas of capillary bed, with correspondingly extensive damage of the blood supply. Our largest individual roentgen portals measured 12 × 16 cm., and 7 of the cases had radium alone, 37 radium and roentgen radiation. Involvement of the ileum for short segments was demonstrated in only 6 out of 44 cases, 2 of ileum alone, and 4 in combination with a segment of large bowel. The ileal damage in all 6 cases was localized in or near the midline, and at the cervical level. In every case the ileum was adherent in the pelvis, to a loop of sigmoid usually, once to the uterus, and once bound down firmly to a pus tube. All ileal lesions were discovered at operation. One could have been discovered on the roentgenograms, where a gas-filled coil of ileum maintained a constant fixed position in relation to strictured sigmoid, but it was missed at the time. In 42 cases, the damage was predominantly in the large bowel, solely in 38, and combined with a short piece of adherent ileum in four. More detailed location of the injury is given in Table II.

TABLE II

LOCATION OF INJURED BOWEL SEGMENTS

Rectum alone	1
Rectum and rectosigmoid junction	3
Rectosigmoid and adjacent distal sigmoid	17
Mid-sigmoid, short segment	3
Proximal sigmoid, short segment	1
Long strip of sigmoid	8
Ileum alone	2
Two or more areas of narrowing (1 area ileum, 1 sigmoid,—4)	9

It will be seen that the large bowel injuries are limited further to the rectum and sigmoid, with the greatest number in the distal sigmoid and at the rectosigmoid junction. The validity of some of the divisions as to distal, mid and proximal is questionable, as the coils shift and turn on one another according to degree of filling and change of positioning in roentgenograms. They are given for an average appearance on a number of roentgenograms, but cannot be considered as arbitrarily fixed. It is obvious from the table why a complete picture of the lesion cannot in many cases be obtained by proctosigmoidoscopy. The instrument passes only to the first point of narrowing. The extent of the stricture, or the presence of a second stricture proximal to the first, can be discovered only by the use of a thin barium stream through the canalized areas.

Our lesions then are predominantly in the midline; all are included in an arc 5 to 6 cm. from the midline and the cervical level, where summation of irradiation effect is obtained with our techniques. Most writers on the subject offer suggestions and individualized techniques for avoiding such injuries, but it is difficult to see how they can ever be completely eradicated as long as we continue to try to administer a dose of 7 or more threshold erythema doses to areas of pelvic cancer. There will always be a few cases with pieces of intestine adherent to the uterus, or bound down close to it against other structures, or gas-distended

FIG. 1. Rectum and distal sigmoid distended by gas, their anterior walls obviously close to the cervix. This confirmed by lateral view, not shown here. Applicators are of the Manchester type. Case from the New York Infirmary for Women and Children.

loops of sigmoid thrusting themselves close against the radium source in the cervix. Two roentgenograms illustrate the last named effect. Figure 1 shows the location of applicators, and adjacent gut distended by gas. Figure 2 shows the resultant injury diagnosed by enema ten months later.

INITIAL TREATMENT PROCEDURES

Our study of this condition has been carried on for seven years. The first cases we discovered were sent for obstruction supposedly due to spread of malignancy. Cases of rectosigmoid irritability with symptoms of diarrhea, bleeding, tenesmus, cramps, and a clinical diagnosis of "irradiation proctitis" were next differentiated. Sometimes a localized narrowing was found, sometimes a more generalized but transient spasm, sometimes where the symptoms were mild, nothing more than a little mucosal hyperemia. For a while we did barium enema and ileal studies on all cases of so-called "irradiation sickness," but this proved unrewarding in proportion to the trouble and expense; it was soon abandoned, and comments on it are outside the scope of this report. Now the Woman's Hospital routine on all cases of cancer of the cervix includes cystoscopic and proctoscopic examinations before any irradiation is given. Several cases of low grade proctitis or cystitis have been found, and irradiation and follow-up is individualized accordingly. Another small but sometimes important item is the avoidance of routine administration of cathartics while the patient is in the hospital for radium treatment, and the forbidding of self-dosage during the ambulatory roentgen course. Too liberal use of milk of magnesia in the hospital was related to severe diarrhea immediately after radium treatment in 7 cases, with epsom salts and cascara implicated in 2 more. To be sure, the cathartic was not the primary case of actual bowel damage, but it was not soothing to an irritable bowel segment.

FIG. 2. Stricture diagnosed ten months later, exactly at level of radium applicators. Both mucosal ulceration and perirectal infiltration were present. Compare with Case 11, Figure 4*A*, which shows similar type of lesion.

Other reflex sources of increased peristalsis, such as gallstones and duodenal ulcer, were present in a few patients, and 2 had lues, though any connection in the latter circumstances is highly questionable. Our system of treatment provides radium first in Stages I, II, and early III (Schmitz' classification); roentgen irradiation first in late Stage III, any badly infected cases, and in Stage IV for palliation with radium used later in Stage IV only if marked improvement occurs from roentgen irradiation; Stage V cases may be treated with either roentgen irradiation or radium, depending on their status, but not both. Where roentgen irradiation has been used first, no immediate bowl difficulty to interfere with subsequent radium has arisen. The reverse has not always been true. Four cases had such severe bowel reactions following radium that the roentgen radiation series could not be given, and in a fifth case, judged by hindsight, it should not have been given.

PATHOLOGICAL AND CLINICAL FEATURES

The primary damage in cases of gross bowel injury appears to be to the smaller branches of the hemorrhoidal vessels supplying the bowel, with thrombotic occlusion thereof resulting in infarction, and mucosal ulceration of the involved segment. Spasm further increases the ischemia so produced, which is not immediate but gradual, as areas of attempted repair are found with the necrosis. Obviously the cellular elements for this reaction must be supplied by some still functioning vessels, and the proportion of these determine the prognosis as to complete restoration, healing by fibrosis and scar, or perforation. The fibrotic changes may involve the bowel only, or may extend to the surrounding tissues with formation of a dense fibrous mass involving the structures of the lower pelvis, simulating the frozen pelvis of massive malignant invasion. Such an event adds greatly to the intrinsic luminal distortion.

The principal feature of the clinical picture is diarrhea, which may occur as early as the third week after beginning of treatment or some months thereafter. This may be a mild disturbance with four to six stools daily containing mucus and blood; or there may be severe rectal pain, tenesmus and "bearing down" sensation in the lower abdomen with the passage of considerable gross blood or clots. With these severe cases, there may be anorexia, nausea, vomiting, and obstipation with abdominal distention, and it is this picture that suggests obstruction to the surgeon. The mechanism is intense edema and severe spasm of the involved intestine followed by a varying degree of fibrosis with luminal contracture which accompanies the localized acute sigmoiditis and perisigmoiditis and its varying degree of ulceration. The latter may be severe enough to result in necrosis of the entire thickness of the bowel wall with resulting leakage into the peritoneum, vagina, or perirectal tissues to complicate the picture.

DIAGNOSIS

Such a development as the above has heretofore often spelled hopeless recurrence to the physician, who has relegated the sufferer to a "home for incurables," where abandonment of all but analgesic therapy has sacrificed the patient. We have infrequently (Table 1) seen neoplastic involvement of the bowel among such cases, some of who have come to operation or autopsy, with practically all of the remainder subject to continuous follow-up to the present. Obstructive symptoms with pain and rectal bleeding months after irradiation should therefore raise the presumption of irradiation effect until proved otherwise. A careful chronological history, roentgen studies by barium enema, sigmoidoscopy, and if possible biopsy should be employed in all cases. Functional bowel disturbance, dietary or cathartic insult, intercurrent intestinal infection and possible malignant disease may be differential alternatives to be considered and ruled out.

PROTOSCOPIC EXAMINATION

The rectal examination and proctoscopic

appearance may be striking. Since the lesions usually occur on the anterior wall of the bowel from 8 to 10 cm. from the anus at approximately the level of the cervix, or in the rectosigmoid or the distal sigmoidal loop, they are often subject to direct visualization. However, edema, spasm, and pain preclude viewing beyond the periphery of the involved area in many cases, and one must rely on roentgen examination.

In the milder reactions hyperemia, spasm, and increased mucous secretion are seen over the area, the membrane appearing like red velvet. There is friability to touch with easy bleeding on manipulation. Bullous edema, severe congestion, and multiple bleeding points may be seen scattered over a beefy red mucosa. When ulceration occurs there may be single or, less often, multiple areas of oval or irregular shape surrounded by a zone of hyperemia, the whole in a pale edematous plaque of indurated bowel wall of varying extent. The ulcer base is covered with grayish slough and the margins may appear somewhat raised and irregular. Its diameter may vary from 1 to 4 cm. Ulcers extending about the whole circumference of the gut as a band 4 cm. wide have been described. The induration will extend on the average over an area roughly double the area of the ulcer.

As healing occurs the slough separates and the ulcer shrinks. In the more caudad lesions on the anterior rectal wall, the eventual appearance is that of a pale, atrophic area the color and surface texture of a pigskin glove, spotted with small telangiectases. A puckered scar may mark the ulcer site, and here an irregular feel as of underlying scar bands will present itself to the examining finger. In the sigmoidal cases the caudal end of the involved area can be visualized as a fawn colored concentric contraction of the bowel. In severe cases with marked fibrosis of the perirectosigmoidal tissues, the rectum seems embedded as in a mould, which prevents instrumentation beyond the rectal ampulla. On palpation, a semilunar firm ridge may be felt at the fingertip extending in a band 1 or 2 cm. broad from one side of the pelvis to the other and representing thickened, contracted, fibrotic parametrial tissue. These are the cases that mimic massive neoplastic invasion of the pelvis. The most marked constrictive phenomena appear in higher lesions.

BARIUM ENEMA EXAMINATION

Types and variations of roentgen findings will be exhibited in the case material and illustrative roentgenograms. The barium enema technique is of diagnostic importance and will be described. Large, full enemata are no longer used, even though the strictured area is wide enough to admit them, first because it is usually easier to let barium run in from an elevated enema can than it is for the patient to expel it past a stricture, and second, because a rectum dilated by barium and barium-filled sigmoid coils overlapping one another may completely conceal a short stricture. We usually start with a pint or less of thin barium suspension, let it run in slowly under roentgenoscopic control, with the patient in the Trendelenburg position, until the stricture is reached, then let barium trickle past it if possible, to outline the extent of the stricture and to rule out a second strictured area higher up. We have never seen stricture as high as the iliac colon and prefer not to fill this part. In stout patients it may be necessary to increase the thickness of the suspension in order to get good shadows. Sometimes the barium runs in so freely that it cannot be completely controlled. Because of the stricture, whether spastic or fibrotic, these patients are often poorly prepared, and mottled gas shadows and irregular filling of this sort must not be confused with the raggedness of spasm or the atypical irregularities of some strictures. As to position for roentgenograms, direct anteroposterior or posteroanterior views are frequently non-diagnostic, and right or left oblique positions are required, depending on the location of the lesion and the position of the sigmoid coils. We have had cases in which the stric-

ture could be demonstrated only in a direct lateral view. It is preferable to make roentgenograms under fluoroscopic control, but at least the proper angles should be selected by fluoroscopy. Post-evacuation roentgenograms are desirable for comparison with the maximum attempted filling.

MEDICAL TREATMENT

Observation leads us to believe that in general this condition is self limited, and, barring certain untoward developments to be later considered, is best treated by conservative and medical means, applied with good judgment and individualization.

Bed rest with bland low residue diet should be employed during the severe symptoms. Since the nutritional state is often poor, an ample vitamin intake is indicated and we feel that this is best provided by appropriate concentrates rather than in the dietary, as fruit and plant substances are too stimulating to the bowel.

Antispasmodics should be given to full effect. Phenobarbital, other barbiturates, and a belladonna preparation in adequate amounts usually suffice and seem more satisfactory than the newer substitutes. For pain, acetyl salicylic acid with acetophenetidine and more powerful barbiturates will answer the purpose in most cases and should be tried before opiates are ordered. The latter may be necessary at times but should not be used routinely as they increase the tendency to distention and may break the compensatory effort of the bowel.

The bowel content should be kept soft to prevent irritating impaction above the involved area by appropriate dosage of liquid petrolatum products. Cathartics are interdicted.

A solution of 1 per cent tannic acid as a 6 ounce retention enema given daily at first, and later on alternate days, has proved useful, especially in limiting bleeding. Monsel's solution has been used for the same purpose. Starch water or mineral oil enemata may also be found soothing.

An amelioration of the edema and spasm may be anticipated with some natural dilatation of the affected lumen by the fecal stream in time, and strict conservative treatment should be persisted in for some weeks after the initial improvement.

If there is frequent vomiting and progressive distention (and it is to be remembered that severe pain in itself will induce vomiting in many individuals) so that real obstruction is feared, preliminary roentgenograms of the abdomen for gas patterns and fluid levels should be made at intervals, since dilatation of the small intestine may occur with obstruction of the sigmoid. The state of body hydration and chemical balance should be observed by determination of the non-protein nitrogen, chlorides, carbon dioxide combining power and hemacrit readings, and any abnormality corrected. Transfusion of whole blood or plasma may be indicated. If evidence of true obstruction exists, a double lumened tube may be passed for decompression and emptying, and for the administration of fluid feedings such as milk, gruel and thick soups. If conservative means fail, this is good preparation for surgical treatment.

SURGICAL INDICATIONS

The application of surgery to these cases requires the greatest judgment. In general these patients are in poor condition and are not good risks. Indications for interference with consequent colostomy should be clear cut and not a matter of extemporization. In the past the following have been cited as calling for operative treatment: (1) obstruction; (2) severe repeated bleeding; (3) severe proctitis and intractable diarrhea calling for diversion of the fecal stream to promote healing; (4) marked weight loss; (5) prevention of perforation of necrotic bowel wall.

Of the 12 operated cases in our material there was only 1 with indisputable evidence of obstruction and this was ileal in origin. In the others with sigmoiditis there was no true evidence of obstruction in any case. Study of the clinical records shows that all either had spontaneous stools or adequate returns from enemata preopera-

tively. At least 8 were operated on in the acute phase of the inflammatory reaction. In some of these cases the surgery was frankly exploratory or for perforation. In several instances the surgeon's notes mention the threatening aspect of the barium enema findings and the striking narrowing of the stricture as indicating operation, but this has been shown to be insufficient evidence to warrant such a decision. Therefore, the conclusively established diagnosis of obstruction has not been a frequent indication for surgery in our experience. If established, its site must be recognized for in a case in our series where multiple lesions were present the lesion responsible for the condition was overlooked with consequent failure and death.

There were 2 postoperative deaths from massive hemorrhage from the bowel. In both, colostomy had been done and was inadequate to prevent the fatal outcome.

Proctitis, diarrhea and weight loss we do not feel indicate operation in the acute phase of the lesion until bed rest and careful conservative measures have failed.

It is difficult to see how one can anticipate perforation of the necrotic bowel before the event. If early perforation has occurred, colostomy alone will not solve the problem.

If after critical study operation seems called for, simple colostomy would appear to be the operation of choice in most cases. Resection with anastomosis has been successfully done, but the danger of anastomosing possibly ischemic bowel should be borne in mind. Abdominoperineal resection is considered an unjustifiably severe procedure except for the rare case. However, in the unusual patient, where repeated large blood loss might threaten an eventual fatal issue from massive hemorrhage, colostomy is obviously inadequate and some form of resection with removal of the damaged intestine is necessary.

CLASSIFICATION

For presentation of case material, some sort of classification is convenient, and we have adopted the following.

Group I. Temporary lesions, showing definite localized narrowing on barium enema and characteristic proctoscopic findings, wherein roentgen evidence of the lesion disappears completely or almost completely in a year or less, clinical subsidence by proctoscopy corresponds, and symptoms are of short duration.

Group II. Lesions rapidly becoming symptomless, in which slight, moderate, or rarely marked degrees of narrowing of the bowel persist longer than a year, and are presumed to be permanent. Here there is limited scarring and contraction without serious impairment of blood supply.

Group III. Severe grades of bowel injury, with enough impairment of blood supply to cause instability, without gross necrosis. Here are found varying degrees of permanent narrowing of the intestine, together with symptoms of bowel discomfort, usually intermittent. Breaks in compensation occur, due to the underlying status plus local factors of improper diet, lowered systemic resistance, remote reflex causes of spasm, and so forth, and a poorly nourished mucosa breaks down readily into ulceration, usually with intervening periods of healing. Occasionally these cases finally pass into Group IV with gross tissue breakdown.

Group IV. The most severe grade of damage, in which there is profound, though often narrowly localized, tissue destruction, with too much loss of blood supply to allow of adequate reparative processes for survival of the tissue. Necrosis, perforation, fistulae, peritonitis and severe hemorrhage are found, and surgical intervention in some form is generally required.

This classification is based on known or estimated degrees of blood supply impairment and tissue destruction plus the fact of outcome, after the passage of a reasonable period of time. It cannot be satisfactorily applied in the fresh case, where it is difficult to foresee the degree of reponse to be expected from treatment. Severity of initial symptoms is not a reliable measure of this. Our cases at the present writing are distributed as follows:

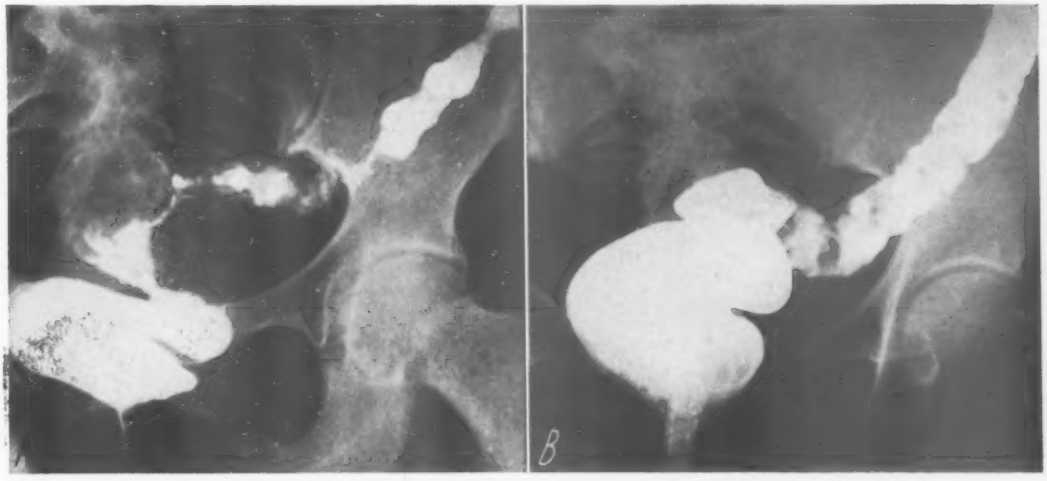

FIG. 3. Case 1. *A*, appearance of bowel in May, 1939, six months after completion of irradiation. *B*, August, 1942. No symptoms since three months after *A* was taken. Normal caliber of overlying coils of sigmoid confirmed by oblique views under fluoroscope. Last proctoscopy in March, 1943, showed normal appearance of bowel.

	Cases
Group I	4
Group II	16
Group III	5
Group IV	12
Early, under one year	7

The largest group is II, and this and Group I, a total of 20 cases, would probably not be included in any series based on severe morbidity of any great duration. We now feel that with our present experience in diagnosis and treatment some of the Group III cases could have been treated and ultimately placed in Group II, and that in a few of the operated cases, operation could have been avoided. Our fourth grouping is therefore ambiguous to the extent that it comprises the operated cases, supposedly representing the lesions of greatest severity, but in several of these the operative indications by our own criteria are now considered debatable. Others show irreparable tissue destruction, with hemorrhage or perforation or both, and the surgical indications are clear cut.

CASE REPORTS

Group I. The Temporary Lesion

This type of case may show initial proctoscopic findings ranging from mild to severe, and almost any degree of bowel deformity by barium enema.

CASE 1. H. A. This patient is interesting because she presented several possibilities of future trouble even before she was given irradiation. She had had a supravaginal hysterectomy and salpingo-oophorectomy for chronic salpingitis seven years before, with multiple adhesions noted at operation. These stump cases offer the hazard of an adherent piece of intestine brought close to the radium source in the cervix, plus the likelihood of diminished screening of even non-adherent bowel by cervical tissue, especially at the point of excision. Also the patient had been admitted to a regular gynecological service, not the cancer division, where she had a curettage with removal of a cervical polyp and cauterization of the cervix. When the polyp was unexpectedly diagnosed malignant, she was treated with a single tube of 100 mg. of radium in the cervix and four 12.5 mg. needles surrounding it for twenty hours, a total of 3,000 mg-hr. This was followed by roentgen therapy, 150 r daily to each of two opposing fields 10 by 16 cm. each and excluding a strip 3 cm. wide in the midline, to a total of 2,000 r (measured in air) on each of four fields over a period of thirty-three days. She had liquid stools on the last four days in the hospital after radium treatment, being given milk of magnesia on the first two of these. After discharge from the hospital, small watery

stools with tenesmus continued for a short time, and a barium enema study was done, which showed moderate spasticity of the whole sigmoid but no definite constriction. Bowel function soon became normal with bland diet and the administration of paregoric and chalk, and the roentgen treatment was begun in two weeks, and concluded without discomfort. Her next complaint, of constipation, gas, and general distress in the lower abdomen, occurred seven months after radium treatment, five and a half months after the end of the roentgen irradiation. Barium enema at this time (Fig. 3*A*) showed a ragged, irregular narrowing of the sigmoid about 11 cm. long. The treatment was again bland diet and paregoric. Bowels were noted as improved after one month, with no symptoms three months later, or at any time in the subsequent four and a half years to date. A stabilized and normal sigmoid is shown in Figure 3*B*, three years after damaged bowel was demonstrated. The patient has been free of cancer for five years.

Group II. Temporary Symptoms, Permanent Constriction

As seen on roentgenograms the usual sequence in this type of lesion is for the area of damage, sometimes involving a considerable length of intestine, to diminish gradually in extent, the ends farthest from the point of maximum irradiation first showing a return to flexibility and normal caliber, until after several years the residual fibrotic constriction may have decreased to 1 cm. or less in length. The greatest improvement comes in the first year and a half, but in a small group we have followed from four to eight years we have observed slow gains in flexibility and widened caliber up to three years, with little change thereafter. Two cases in this group will be shown, the first presenting spastic, edematous, ulcerating gut with no significant evidence of perisigmoidal reaction. The second shows

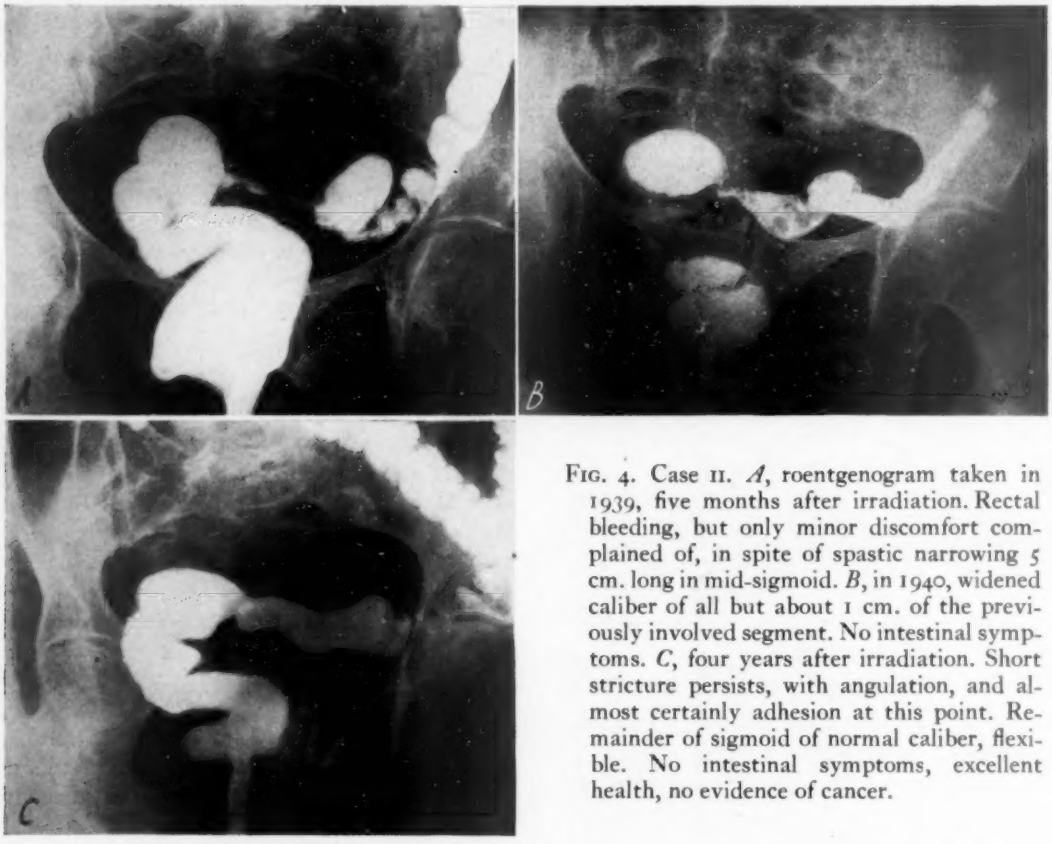

FIG. 4. Case 11. *A*, roentgenogram taken in 1939, five months after irradiation. Rectal bleeding, but only minor discomfort complained of, in spite of spastic narrowing 5 cm. long in mid-sigmoid. *B*, in 1940, widened caliber of all but about 1 cm. of the previously involved segment. No intestinal symptoms. *C*, four years after irradiation. Short stricture persists, with angulation, and almost certainly adhesion at this point. Remainder of sigmoid of normal caliber, flexible. No intestinal symptoms, excellent health, no evidence of cancer.

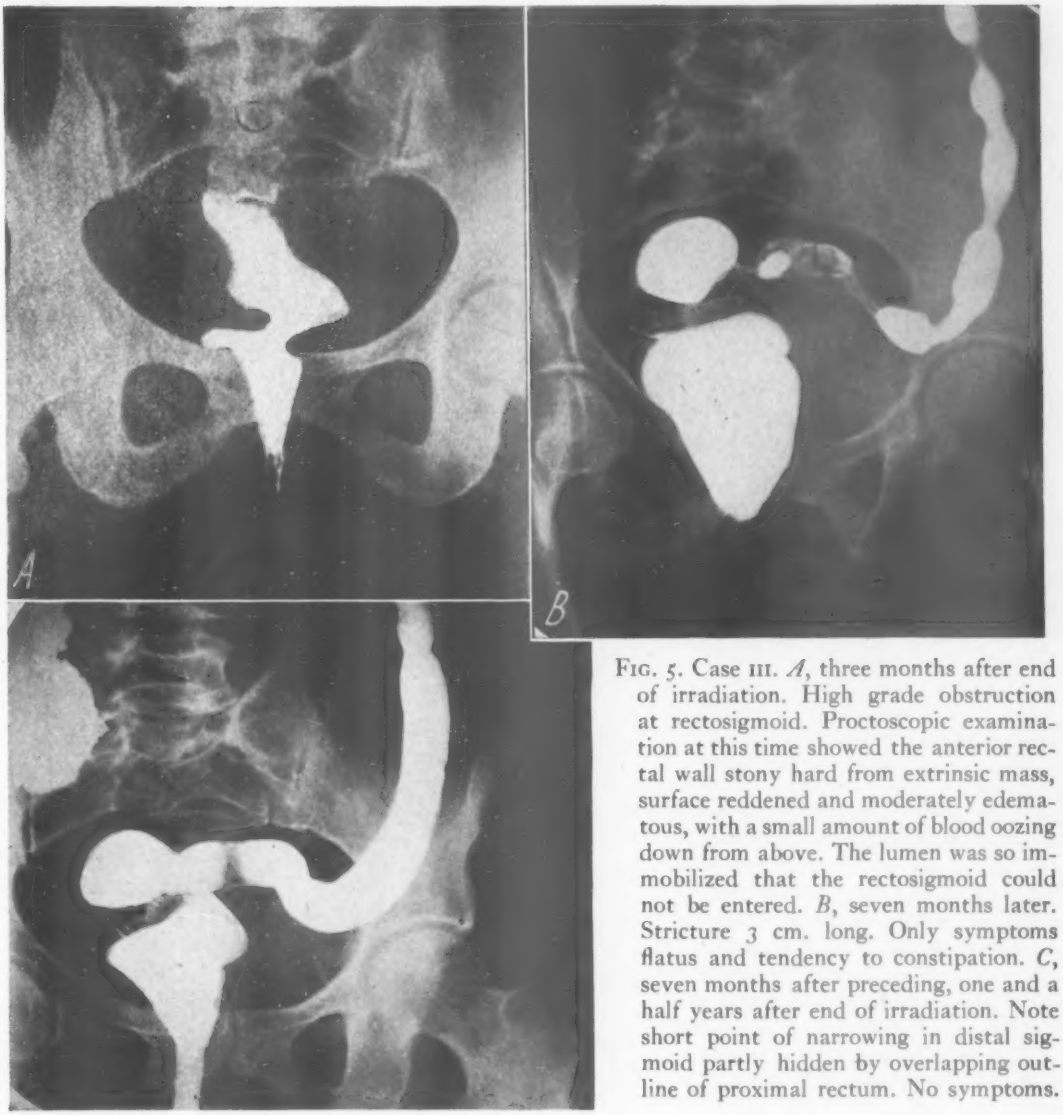

FIG. 5. Case III. *A*, three months after end of irradiation. High grade obstruction at rectosigmoid. Proctoscopic examination at this time showed the anterior rectal wall stony hard from extrinsic mass, surface reddened and moderately edematous, with a small amount of blood oozing down from above. The lumen was so immobilized that the rectosigmoid could not be entered. *B*, seven months later. Stricture 3 cm. long. Only symptoms flatus and tendency to constipation. *C*, seven months after preceding, one and a half years after end of irradiation. Note short point of narrowing in distal sigmoid partly hidden by overlapping outline of proximal rectum. No symptoms.

the typical "extrinsic" lesion described by Todd and others; though we do not consider the distinction between "intrinsic" and "extrinsic" of basic significance, except that the latter must be differentiated from the frozen pelvis of malignancy.

CASE II. B. E. This patient received the maximum irradiation usually given at the Woman's Hospital—6,000 mg-hr. of radium in the form of 100 mg. intracavitary and 6 needles totaling 75 mg. interstitially for twenty-four hours, followed in twelve days by the Ward colpostat using 75 mg. for twenty-four hours; roentgen irradiation consisting of totals of 1,800 r on each of two anterior and two posterior pelvic fields, and 1,4000 r on each of two lateral fields, a total of 10,000 r (measured in air) given to the pelvis. She had advanced disease when first seen, Schmitz Stage III, League of Nations' Stage III, squamous carcinoma, Grade 3, anaplastic. With all this, she seemed surprisingly spry and vigorous, whence an attempt at cure rather than palliation. While in the hospital she was another temporary casualty of the milk of magnesia routine with brief diarrhea which responded to paregoric and chalk, and with no subsequent complaint during roentgen irradiation or up to five months after the end of roentgen therapy and about seven months after

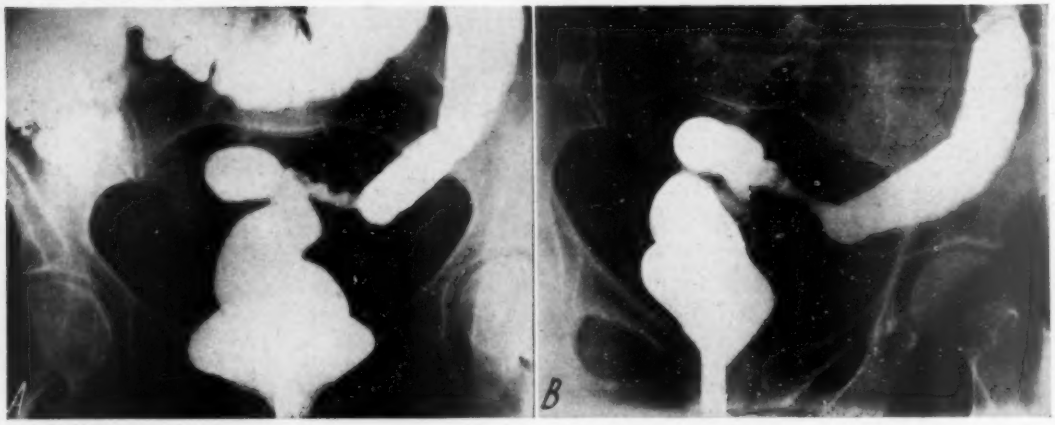

FIG. 6. Case IV. *A*, nine months after irradiation. Patient has recently recovered from an episode of bloody diarrhea. *B*, three years after irradiation. No acute symptoms at this time.

radium treatment. At this latter time, her clinic chart notes "occasional bleeding from rectum though bowels soft and regular with the aid of mineral oil." She was sent for barium enema study, which presented an appearance entirely disproportionate to her symptoms (Fig. 4*A*). She was put on a bland diet and the last sign of bleeding was a month later. Figures 4*B* and *C* show subsequent progress. She has had no further intestinal complaints for the past five years and there is no sign of cancer.

CASE III. M. E. This patient shows a different type of lesion and much more intestinal disturbance, though she had less radium, a total of 4,200 mg-hr. and less roentgen irradiation, 8,000 r (measured in air) to the pelvis, than the preceding case. She was readmitted to the hospital for obstruction three months after the end of irradiation, with symptoms of abdominal pain, distention, nausea, vomiting, and rectal bleeding. Perforation of the sigmoid from post-irradiation necrosis was considered by the surgeon. A cautious barium enema study was made with results shown in Figure 5*A*. She improved quickly on bed rest, low residue diet, mineral oil and sedatives, and operation was not required. After discharge from the hospital, the remaining symptoms gradually disappeared, except for gas and a tendency to constipation, for which she was kept on a bland diet with fruit juice for a considerable time. When seen recently, three years after irradiation, she had no complaints and was clinically free of cancer. The roentgenograms taken after barium enema are shown in Figures 5*B* and *C*. The clinical picture in this case includes features which some consider criteria for surgical intervention and under other circumstances might have been so treated.

Group III. Permanent Stricture, Residual Symptoms

This group is interesting because there are usually intermittent complaints alternating with symptom-free periods.

CASE IV. R. O. (From the New York Infirmary for Women and Children.) The patient had advanced carcinoma of the cervix, League of Nations Stage III. The left half of the cervix was stony hard, the left fornix obliterated, the cervical canal obstructed so that an intracavitary applicator could not be used. She was treated with a radium bomb delivering 1,500 mc-hr. in one hour eighteen minutes, and radon needles delivering 2,200 mc-hr. in thirty-six hours. The roentgen treatment was given over two fields daily with amounts ranging from 200 to 50 r each, the dosage being varied because of intestinal irritability, and a total of 2,000 r to each of four pelvic fields in thirty-eight days. The sequence was the radium bomb, part of roentgen cycle, radon needles, remainder of roentgen irradiation. Treatment was given in January and February, 1938. There was moderate intermittent diarrhea during irradiation and for several months thereafter, followed by a symptomless period, and then a sharp attack of bloody diarrhea six months after treatment. She had two such episodes in 1938, two in 1939; since then she has had constipation and cramps several times a year without bleeding. Her only other complaint during a four year period was the necessity for a peculiar bowel

routine consisting of morning sessions of repeated small evacuations, the whole performance lasting about an hour. (We have seen another case recently with stricture following the same scheme of six to ten small evacuations each morning taking one to two hours.) She took mineral oil, and by her own choice small doses of milk of magnesia. The patient remained clinically free of carcinoma for five and one-half years, until her death late in 1943 of a cardiovascular accident. Four barium enema studies were done in 1938, 1939, 1941 and 1942. Figure *6A* shows the lesion late in 1938, Figure *6B* shows it two and a half years later in 1941. The narrowest portion is no wider, but the length of the narrowed strip has decreased slightly, and this tendency is apparent also in the last barium enema examination made in 1942.

Group IV. Severe Damage, Requiring Operation

While we believe that unjustified surgical interference has occurred in cases of stricture, unavoidable indications for operation do arise. This is more likely to happen in the occasional late case where medical treatment has failed, the failure being based on irreparable bowel damage and evidenced by unremitting severity of symptoms of pain, diarrhea and bleeding, or by signs suggestive of perforation.

CASE V. L. E. This patient for over five years constituted a typical example, though unrecognized, of Group III. During that time she had intermittent attacks of pain, constipation and blood-streaked stools, occurring three or four times a year, with intervals of normal bowel state and excellent health. She had had radium treatment in 1933 for cervical carcinoma, but no roentgen therapy because of marked telangiectasis of the lower abdominal skin and dense pelvic fibrosis following an earlier course of roentgen therapy for fibroids. As we were unaware of the likelihood of sigmoid stricture in her case, no investigation was made, and the attacks were believed due to indiscretions in diet plus hemorrhoids. Finally an unusually severe attack of obstipation and pain occurred, barium enema examination was done (Fig. 7), and the patient was operated upon. Ten centimeters of intestine, including the proximal narrowing seen on the roentgenogram, was resected with end-to-end anastomosis, and a temporary colostomy done, which was closed ten days later. The wall of the intestine was 1½ inches thick at its most constricted part, densely adherent to the uterine fundus and left adnexa, and perforated at its point of attachment. Generalized pelvic fibrosis was present. The affected bowel showed the whitish, rubber tube appearance characteristic of these fibrotic, ischemic lesions. The bowels moved well almost immediately after operation, and the patient had no further difficulty for over a year, when she again complained of severe constipation, and a proctoscopic examination was done. The instrument could not be passed more than 5 inches, where the bowel was narrowed and edematous, but no ulceration was seen. The diagnosis was stricture at the rectosigmoid, and it was assumed that the second narrowing had not been found at operation because of dense adhesions, and that the distal point of excision ran through the section of relatively normal bowel lying between two strictures. Treatment of the lower stricture was proposed, but about this time the patient developed extensive remote metastases in lungs, bone and spinal cord, and died shortly after. At autopsy no malignant disease was found in the pelvis.

FIG. 7. Case v. Note narrowings in mid-sigmoid and distal sigmoid. Actual condition unrecognized for over five years after irradiation, though bowel symptoms had occurred intermittently throughout this period. This whole segment of intestine found at operation to be enmeshed in dense pelvic adhesions. Resection of 10 cm. of bowel including the proximal stricture was done, with end-to-end anastomosis, and a good functional result obtained immediately. Distal narrowing not identified at operation and gave trouble one year later when bowel disturbance recurred.

FIG. 8. Case VI. Colon filled with ingested barium. Not a barium enema. Note narrow stricture of mid-sigmoid, plus ragged outline of distal sigmoid and rectum corresponding to multiple rectal ulcers. (Blebs of gas also present but irrelevant.) The upper stricture caused the immediate obstructive symptoms, precipitated by the weight of barium above the narrowing, but the patient eventually died of hemorrhage from the rectal ulcers.

Her death occurred eight years after the diagnosis and treatment of the pelvic malignancy, and about a year and a half after operation.

Miscellaneous Cases

CASE VI. W. A. This case is reported briefly to show the most extensive damage we have encountered, and to emphasize a caution in the handling of post-irradiation diarrheas. Symptoms appeared five months after radium treatment, three months after the end of roentgen irradiation, and consisted of severe griping pain, bright red blood, and fifteen to twenty stools a day. She was given by her home physician 2 ounces of barium sulfate daily, apparently to lessen the intestinal irritability. After five or six days of this she was brought to the Woman's Hospital with obstipation and great pain, and the bowel condition shown in Figure 8. This is not a barium enema, but an accumulation of ingested barium in the colon above the stricture, and the weight and bulk of the barium evidently served to precipitate a high degree of obstruction. Proctoscopy showed constriction 13 cm. from the anus. There was edema of the mucous membrane. Ulcers about 1 cm. in diameter were scattered at varicus points on the rectal wall, and ulcer patches could be seen above the constriction. A colostomy was performed and the patient did fairly well for a while, but the ulcers did not heal and she died of secondary hemorrhage thirty-four days after operation.

It is obvious that such a mass of barium should never be given by mouth in the presence of low stricture. It is a good principle to avoid any oral medication that could do harm, either bulky material like this, which tends also to dry out in the distal colon, or irritating substances like cathartics, until the nature of the condition is ascertained. As to treatment, this is one of the very rare cases in which permanent colostomy plus abdominoperineal resection might have saved the patient, as the procedure done was no guarantee against the eventual fatal hemorrhage.

CASE VII. D. W. This case is an example of healing of damaged bowel by diverting the fecal stream by colostomy for a period of a year, after which the colostomy was closed and the distal segment allowed to function. The patient was irradiated in May and June, 1937, and developed an acute episode of severe pain, diarrhea and rectal bleeding six months later. In a barium enema examination, not shown here, two narrowings, in the proximal sigmoid and at the rectosigmoid junction, were found. She was treated by bland diet, belladonna and bed rest, with good results. One and a half years later, two years after irradiation, she had another upset, with obstructive symptoms (Fig. 9*A*). The recurrence of symptoms after so long a time suggested severely damaged intestine. Operation was decided upon and a Mikulicz colostomy was done. Many pelvic adhesions were present. The lower 4 inches of the sigmoid and part of the rectum showed white, scarred areas with telangiectatic spots, but not the wide damage seen, for example, in Case V. There were no enlarged lymph nodes, no evidence of metastasis. The colostomy functioned well and was closed after one year (Fig. 9*B*). She was followed for two years more, five years in all. She remained in good health, free from cancer, and with no further bowel distress. She has recently moved to Trinidad, from where she reports by letter.

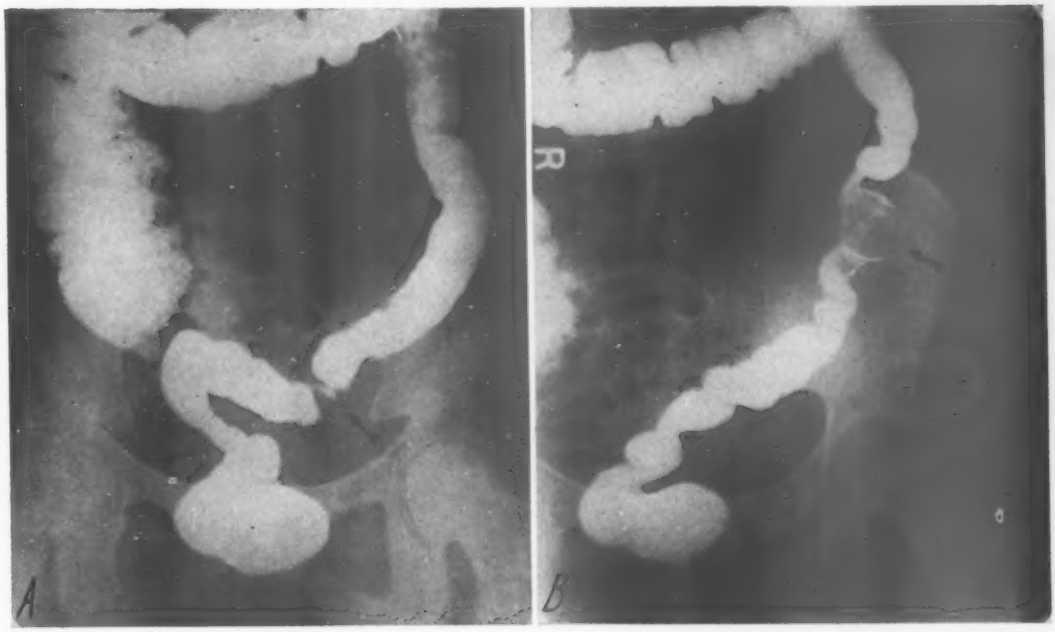

Fig. 9. Case vii. *A*, two years after irradiation. (An earlier barium enema examination described in text.) Although the two strictures seen are not very narrow, there were symptoms suggestive of partial obstruction. As the patient had had a similar severe episode one and a half years before, the state of nutrition of the involved intestine and the extent of the actual damage were considered too uncertain to risk a further waiting policy, and exploratory laparotomy and colostomy were done. *B*, one year later, following closure of colostomy. There are still points of indentation (limited scarring) at the sites of the former strictures, but oblique views under the fluoroscope demonstrate a good degree of flexibility. Note pouching in the descending colon at the point where the colostomy was closed.

Maximum Narrowing Without Symptoms

Case viii. S. M. This case shows how narrow a stricture may exist with relatively normal bowel function. This patient was irradiated in April and May, 1939, and began to have intestinal symptoms six months later, these continuing for about a year and a half. A series of proctoscopies showed characteristic findings, at first edema of the anterior rectal wall, which bled to touch, and later induration only with mucosa friable. She received medical treatment continuously during the period of disturbance, and has been followed in the proctoscopic clinic up to the present. A barium enema examination was made in March, 1943 (Fig. 10) when she had been symptomless for over a year. She has been clinically free of cancer for nearly five years. It seems remarkable that anything can go through such a narrow stricture.

Fig. 10. Case viii. This barium enema examination was made four years after irradiation. Patient has had no intestinal complaints for over a year and actually has normal bowel function. Diverticula seen in sigmoid. This is our case of narrowest symptomless stricture.

FIG. 11. Case IX. Barium enema examination made in October, 1942. The radium responsible for the stricture was given in 1923, 1924, 1925. No roentgen therapy. The patient was symptomless during most of this long interval.

Stricture Discovered Twenty Years After First Irradiation

CASE XI. S. C. Patient was first irradiated for cervical cancer in 1923, and several times in the next two years radium needles were used for small recurrences. Although she had rectal pain and bleeding in July and October, 1924, no examination of the bowel was made and apparently little attention was paid to the complaint then or later. A view of her long follow-up history shows an attack of cramps, vomiting and lower abdominal pain in 1938, a bout of constipation in 1939, and a short spell of rectal bleeding in 1941. It appears likely that earlier episodes were not recorded. She came finally to the gastroenterological clinic in 1942 for abdominal discomfort and distention after eating. A barium enema examination (Fig. 11) shows a strip of narrowing about 6 cm. long in the proximal and mid-sigmoid. Proctoscopic examination showed telangiectasis of the anterior rectal wall. The instrument could not be passed high enough to reach the stricture. The patient was treated by sedatives and attention to diet, and symptoms abated. In a second barium enema examination made five months later with the patient symptomless, the stricture remained unchanged. We are uncertain whether it had anything to do with the indigestion reported five months before. As we have no earlier enema studies it is impossible to know the age of the stricture, but it seems reasonable to date it from her first reported intestinal upset in 1924.

CONCLUSIONS

1. Post-irradiation proctosigmoiditis with stricture is a not uncommon cause of recurrent bowel symptoms after treatment for cancer of the cervix uteri.

2. Such symptoms are rarely due to neoplastic extension, and should be considered a post-irradiation effect until proved otherwise.

3. Severe and extensive post-irradiation damage of the bowel may and often does undergo healing with very little functional impairment, though marked anatomic defect may be present.

4. Such a favorable outcome is materially assisted by careful medical treatment.

5. Surgery should be used only after a critical study of the case, and for definite indications which should be satisfied by the operative procedure selected.

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New York, N. Y.

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Vol. 52, No. 6

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

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Forty-fifth Annual Meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Sept. 18–21, 1945.

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Twenty-eighth Annual Meeting: 1944, to be announced.

EDITORIAL

ABDOMINAL ANEURYSMS

THE importance of abdominal aneurysms as the cause of abdominal pain is not too generally appreciated. The predominant complaint in patients suffering from abdominal aneurysm is pain, which may be sudden and which occurs usually in the left side of the epigastrium, no doubt due to the fact that the greatest number of abdominal aneurysms are found in the upper third of the abdominal aorta. The pain is usually of a progressive nature and increases in severity with the progress of the disease. The pain radiates to the back in the region of the upper lumbar spine and may be associated with a shooting radiating type of pain to the left buttock, left lower quadrant region and occasionally to the entire left extremity, simulating a sciatic neuritis. The pain has been variously described as boring or pounding, and patients have often been able to obtain relief by changing from a supine to a prone position. One peculiarity of the pain is that it is unassociated with the intake of food, though gastrointestinal symptoms may often be complained of when the stomach, duodenum and colon adjacent to the aneurysm become involved by pressure. In so-called silent areas aneurysms may attain considerable size and remain asymptomatic, while in other, more critical sites, severe manifestations appear early and the symptoms which arise in the gastrointestinal, genitourinary or biliary tracts are usually the result of pressure or rupture of the aneurysm.

Quite recently Scott,[1] in a report of 96 cases of abdominal aneurysms, has called attention to the fact that the rôle of syphilis in the etiology of aneurysms of the thoracic aorta has been repeatedly and conclusively demonstrated, whereas aneurysms of the abdominal aorta have been less thoroughly studied. In most previous reports aneurysms occurring in each of the various abdominal arteries, namely the aortic, hepatic, splenic, etc., have usually been considered in separate communications and under these circumstances attention has not been directed toward similar and dissimilar features of the group as a whole. He points out that with the possible exception of aneurysms of the renal artery, syphilis, arteriosclerosis and bacterial agents are the important etiological factors. In the renal artery most observers agree that trauma is the most important cause. In the abdominal aorta the majority of syphilitic aneurysms arise from the proximal portion, above the origin of the renal arteries, and from branches in its upper portion, namely, the celiac axis and the superior mesenteric artery, and when syphilitic aneurysms occur in these branches, they almost invariably arise at or near their point of origin. Arteriosclerotic aneurysms arise in the distal portion of the abdominal aorta below its bifurcation or in the distal portion of the splenic artery. Mycotic aneurysms are rare in the abdominal aorta itself but are common and fairly equally distributed among its branches.

Scott points out that a fairly sharp delineation exists between the patients with syphilitic and arteriosclerotic aneurysms in his group of cases by age alone. Seventy-five per cent of the syphilitic aneurysms in his cases occurred in patients under the age of fifty, whereas all of the arteriosclerotic aneurysms occurred after this age. No

[1] Scott, Virgil. Abdominal aneurysms. *Am. J. Syph., Gonor. & Ven. Dis.*, Nov., 1944, *28*, 682–710.

syphilitic aneurysms were observed after the age of seventy.

The relatively high percentage of mycotic aneurysms in the younger age groups is related to the incidence of subacute bacterial endocarditis.

Hubeny and Pollack[2] point out the importance of syphilis in the etiology of aneurysm occurring in patients under the age of sixty and they call attention to arteriosclerosis in the older age groups, and most writers have agreed that arteriosclerosis is the major cause of aneurysms in elderly patients.

The variability of the signs and symptoms of abdominal aneurysm is dependent upon the site of the aneurysm itself and the involved adjacent structures.

Roentgenologic studies may be of considerable importance in determining the presence of abdominal aneurysm. The classical sign of erosion of the vertebral column resulting from pressure of the aneurysm is a well known sign, but that calcium deposits in the wall of the aneurysmal mass may be demonstrated by roentgen study is not too generally appreciated. The demonstration of the calcium plaques by lateral and oblique roentgen studies of the abdomen in severe unexplained abdominal pain may be of considerable diagnostic aid and such a study should not be neglected. The severe pain associated with abdominal aneurysm simulates in many cases the syndrome of rupture of an abdominal hollow viscus, and when the roentgen signs are absent in these suspected cases of ruptured hollow viscera the presence of an abdominal aneurysm should be considered and roentgenological studies made with this in mind.

[2] Hubeny, M. J., and Pollack, S. Saccular abdominal aortic aneurysm. Am. J. Roentgenol. & Rad. Therapy, 1940, *43*, 385–393.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Sept. 18–21, 1945.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: 1945, to be announced.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. F. H. Squire, 1754 W. Congress St., Chicago 12, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meets annually second Sunday in May.

IOWA X-RAY CLUB
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. R. Brindle, 501 Grand Ave., Asbury Pk. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. An-

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

nual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

North Dakota Radiological Society
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. Henry Snow, 1061 Reibold Bldg., Dayton, Ohio. Meets during annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. R. P. Barden, University Hospital, Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. L. M. J. Freedman, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Murray P. George, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary Dr. A.M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. E. W. Spinzig, 2646 Potomac, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Diego Roentgen Society
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Martha Mottram, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Toland Hall, University of California Hospital, second six months at Lane Hall, Stanford University Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. Asa E. Seeds, Baylor Hospital, Dallas, Texas. Next annual meeting, Temple, Texas, Jan. 17, 1945.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly, third Thursday evening.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Continental Europe

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

THE IMPORTANCE OF HISTORICAL REFRESHMENT

TO THE EDITOR:

The "refresher course" has long since become a distinct contribution to the educational value of our annual meetings. By this means we are enabled to correlate the established fundamentals of our past radiologic education with the expanding requirements of modern practice. This necessary adaptation may be materially furthered by occasional survey of the hard-won accomplishments of our vanguard pioneers.

In the October number of THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY there appears "A Historical Note on Roentgenologic Literature" from the pen of our recent President, Dr. Sherwood Moore. In this communication he writes especially of *The American X-ray Journal*, that literary forerunner of our present thriving Journal, and of Dr. Heber Robarts, its Editor and the first President of the American Roentgen Ray Society. Dr. Moore observes that ". . . many, if not most, radiologists are unaware of the existence of this publication important in its day." True words indeed, but nevertheless a sad commentary on the efforts of more than one historian who has striven to revive the memory of these fast-fading figures of Radiology's heroic Past. The resultant records lie freely open within our libraries for all to see, and Dr. Moore does well to take official note of the degree of general ignorance that can be engendered only by unthinking disregard of them.

Such of these records, and they are by no means meagre, as apply to the career of Heber Robarts, reveal not only the vicissitudes of the pioneer, but also the physical distresses of the true martyr to Science through the effect of radiation damage. For him, the advent of death was inevitable as the result of his devotion to radium therapy, but his constitutional strength grievously extended his years of suffering.

The quality of self-sacrifice, as manifested by the lives of Robarts and those like him, is inseparable from that revealed in conflict under our Flag. To contemplate such lives, even in tardy review, is good for the soul.

PERCY BROWN

Vol. 52, No. 6

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 103 Rutledge Ave.
Charleston, S. C.

A NEW ANTEROPOSTERIOR PROJECTION FOR ROENTGENOGRAPHY OF THE OPTIC FORAMEN

By SIDNEY ALEXANDER, R.T.

From the Department of Internal Medicine, New York State Psychiatric Institute and Hospital
NEW YORK, NEW YORK

A REVIEW of the literature for the past fifteen years reveals a number of methods for roentgenographing the optic foramen, all of which utilize the posteroanterior position. In the Rhese method,[8] the head is placed in the forehead-nose position and then rotated obliquely to either side, the chin being in contact with the cassette or table top. The central ray passes through a point 2½ inches above and behind the opposite external auditory canal, directly though the orbit near the film. Pfeiffer[13] objects to this method of projecting the ethmoid sinuses through the margin of the orbit, as a hit and miss affair. He describes his own "V" tunnel apparatus that produces good results without changing the original position of the head. Instead of rotating the head, the tube is moved from side to side, and the cassette is changed from one side of the tunnel to the other.

The MacMillian special position is similar to the Rhese position, except that a 15° angle board is used and the central ray is projected vertically so that it passes through the head from the opposite occipital region and emerges at the orbit. This technique is simplified by the use of a Camp[2] localizer. Epstein and Kulick[4] and Wittenberg[18] describe a simple adaptation of the Bullitt mastoid apparatus for roentgenographing the optic foramen. More recently, Epstein and Kulick[5] presented a method in which the use of mechanical devices for positioning the head are dispensed with. The method is similar to the MacMillian position, the patient being prone with the malar eminence, tip of nose, and the superior orbital ridge touching the cassette. However,

Fig. 1. The anteroposterior position for the optic foramen, including the use of a 40° cardboard triangle.

they suggest elevating the superior orbital ridge 1 inch, by means of a wedge of felt, to prevent foreshortening of the optic foramen in the longitudinal axis. Instead of the horizontal position, a 15° angle board may be used, similar to that prescribed in the MacMillian position.

A study was carried out using the anteroposterior position for roentgenographing the optic foramen, the results of which are recorded in this paper.

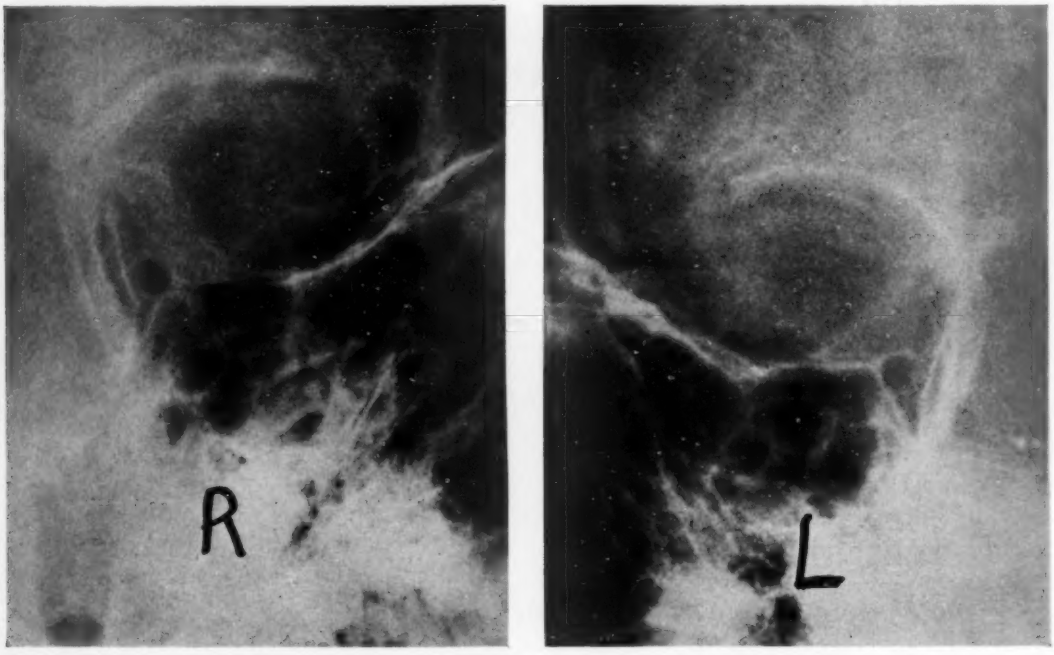

Fig. 2. An anteroposterior projection of both foramina.

PROCEDURE

The patient is supine. An angle board of 15° reversed is used and the patient lies with his head extended overhanging the angle board. The head is rotated 40°, either to the left or right, depending upon the optic foramen to be projected, i.e., left for the right foramen and right for the left. The proper rotation is obtained by means of a cardboard triangle with a 40° angle. This triangle is placed on the horizontal table in back of the skull, and the head is rotated until the mid-anteroposterior line of the skull makes a 40° angle with the horizontal table. The tube is centered so that the central ray is perpendicular to the horizontal table and passes through the orbit which is being roentgenographed (Fig. 1). Technical factors are as follows: small focal spot, anode film distance 30 inches, 55 kv. and 15 ma., at ½ second with a small extension cone.

DISCUSSION

Various other anteroposterior positions were tried before the final procedure was adopted. With the skull placed horizontally and rotated from 10° to 80° in either direction, it was ascertained that the optic foramen could be visualized at 30° and 35°. However, the projections were not satisfactory since the shadow of the mastoid and sinuses overlapped that of the optic foramen. With a 35° rotation the foramen fell within the orbit while at 30° it was outside. It was noted, too, that the head had to be rotated in the opposite direction of the foramen to be roentgenographed, i.e., to the left when the right foramen is desired, and vice

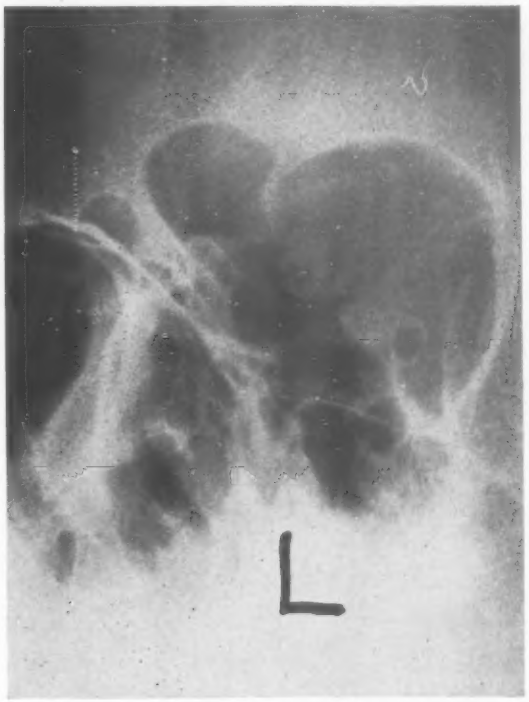

Fig. 3. A posteroanterior projection (MacMillian position) of the optic foramen of the same patient for comparison.

versa. Angle boards from 5° to 25°, placed so as to flex the head, were tried. The most satisfactory results were obtained with an angle board of 15° and a 40° rotation of the head. There was still, however, some overlapping shadows with noticeable enlargement of the foramen as compared with the posteroanterior MacMillian position. A 5° tilt, both toward the nose and toward the occipital region, produced no changes.

The final step is that described in the procedure of this paper. This anteroposterior projection compares favorably with the procedures in which the posteroanterior position is employed. It is a simple procedure as far as both the technician and patient are concerned. It involves no costly or additional equipment. A view of the foramen is obtained clear of the mastoid and sinuses (Fig. 2). Although a detailed comparison of both the anteroposterior and posteroanterior methods, by actual measurements of the optic foramen, has not been attempted at this time, in general the foramina appear very similar by both methods (Fig. 3). The procedure is not only simple and easily carried out by the roentgen technician, but it is also much easier for the patient, both adults and children. The results obtained compare satisfactorily with those obtained by the older methods.

SUMMARY

1. A method using the anteroposterior position for roentgenographing the optic foramen is presented.

2. The method is simple and easy for both technician and patient.

3. The results can be duplicated and are as satisfactory as those obtained by the posteroanterior methods in use at present.

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